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SECTION OF PEDIATRICS

Lower Section Room, Municipal Auditorium, Springfield, Wednesday, June 10, 1936

PRESIDING:
Dr. George P. Hunt, Pittsfield, Chairman.
Dr. James M. Baty, Belmont and Boston, Secretary.

Panel Discussion on "Rheumatism and Rheumatic Heart Disease in Early Life."

Dr. John Lovett Morse, Boston, Leader
Dr. Eli Friedman, Boston
Dr. Hyman Green, Boston
Dr. T. Duckett Jones, Boston
Dr. Tracy B. Mallory, Boston
Dr. Oliver H. Stansfield, Worcester
Dr. Paul D. White, Boston.

CHAIRMAN HUNT: *Members of the Section of Pediatrics*—Your officers this year believed that possibly to the general practitioners this sort of discussion would be most interesting, instructive and helpful. Through the kindness of the leader, it has been possible to arrange for this meeting this group of gentlemen who have put a lot of thought, effort and study into the subject of rheumatism and rheumatic heart disease in early life. We are particularly fortunate in attempting, at least, to cover the etiology, pathology, symptomatology, treatment and prognosis; and we are going to close promptly at 11.30 so that the members can attend the Annual Meeting at the Hotel Kimball.

It is an extreme pleasure to me as Chairman of your Section this year to present to you these men: Dr. Paul D. White of Boston, who will speak about the electrocardiogram; Dr. Tracy B. Mallory of Boston, the pathology; Dr. T. Duckett Jones, the etiology; and the other members, Dr. Eli Friedman, Dr. Hyman Green, and Dr. Stansfield who will come into the discussion. We want, particularly the men who have given their time and effort to come down here for this meeting, a very free discussion, and, to simplify matters, I am asking members who have questions if they will please write them and pass them up to the platform so that they may be discussed and answered by the panel. On the other hand, if there are questions which come up on the floor, I hope that members will come to the platform and use the microphone so that every word of the questions, discussion and answers may be heard by everybody here.

It is a great pleasure to present to this Section again Dr. John Lovett Morse, who will lead this discussion in "Rheumatism and Rheumatic Heart Disease in Early Life." Dr. Morse.

DR. JOHN LOVETT MORSE: Mr. Chairman, the object of this panel is to clarify our views as to rheumatism and rheumatic heart disease in early life. We are going to limit ourselves to rheumatism and acute, or active, rheumatic heart disease and will not consider chronic valvular disease or late cardiac failure.

I am going to treat these gentlemen as though they were my students and ask them questions, and I hope I can ask them some they can't answer. I hope that they will disagree with each other, but I am afraid they won't. I am afraid they all think alike. As to questions from the floor, if you will write them out and send them up here, we will try to answer them.

Now a few words as to the etiology. A few years ago the question as to the etiology of rheumatism and rheumatic heart disease was apparently solved. It was quite generally accepted that they were due to the *Streptococcus hemolyticus* and allergy. We are not so certain now. Are they caused by a virus? I am always a little skeptical about attributing a disease to a virus, because it seems to me that the tendency is now, when we do not know what the cause of a disease is, to take refuge in a virus, the same as we used to in miasma and malaria.

Dr. Jones is going to discuss the etiology for us. There are a few questions which I have written down here which I hope he will take up. First, the importance of environment and economic status. Is there a familial susceptibility to the disease? If so, does this mean that it is contagious? Is a vitamin C deficiency a necessary background? Does a subclinical degree of scurvy constitute the rheumatic tendency? Is there a chemical imbalance, "acid diathesis", according to Payne, which is the cause of rheumatism or which increases susceptibility to it? That sounds to me like quack medicine. What is the relation of upper respiratory infections to recurrent attacks? Dr. Jones.

DR. T. DUCKETT JONES: *Mr. Chairman, Ladies and Gentlemen*—I regret that it will not be possible to answer all of Dr. Morse's questions because of our present lack of knowledge. I shall attempt to review briefly some of the considerations which appear pertinent to the etiology of rheumatic fever. Many of the statements will be quite general and critical, and minute details are impossible in the short time at my disposal. These may be supplied in the discussion should those present desire them.

It will be wise to state at the outset that we do not know the etiology of rheumatic fever, and, in so far as I am aware, no one has ever reproduced the disease process in animals. Thus, Koch's postulates have never been fulfilled. For the past forty years an almost unlimited number of data have appeared in medical literature with various claims as to the streptococcus as a cause of rheumatic fever. I will, therefore, deal largely with this phase of the problem.

The first suggestion was that rheumatic fever was the result of a generalized streptococcus bacteremia. This was suggested a few years before

the work of Poynton and Paine (1900), but these workers gave the greatest stimulus to the probability. As you may recall, these observers claimed to have isolated the agent responsible for rheumatic fever and, in fact, called their organism the "Diplococcus rheumaticus." A large volume of blood culture and postmortem culture work followed during the first quarter of this century. One could cite a list of distinguished investigators who disagreed completely concerning the results of both of these methods of bacteriologic investigation. The value of postmortem cultures has been long questioned, but it remained for Epstein and Kugel (1929) to demonstrate conclusively that their significance is indeed questionable. Many controversial reports appeared before Wilson (1933) and Callow (1933) indicated that blood culture work was not pertinent to the solution of the rheumatic fever problem. Both workers showed that one obtains a high percentage of positive cultures in any acutely ill patient and, further, that normal individuals with respiratory infection had as high a percentage of positive cultures as rheumatic fever subjects. This work is another instance of proper control studies resulting in a more rational evaluation of results, and it does much to discourage the straight bacteremia theory. In the reports of various investigators, experimental animal studies may be seen to have resulted in suppurative joint lesions and bacterial endocardial lesions. Neither of these conditions is met with in human rheumatic fever.

Small (1927) obtained an anhemolytic streptococcus from the blood of a rheumatic fever patient. Injection of saline extracts of this organism resulted in a twenty-four-hour skin reaction in rheumatic fever subjects. Small concluded that this organism was a toxin producer and rheumatic fever the result of this toxin. He called this organism the "streptococcus cardio-arthritidis". Birkhaug likewise made such a suggestion shortly after that of Small. The therapeutic use of antisera and vaccines suggested by Small did not prove of value in the hands of other observers. Some of the pertinent criticism of this theory will be alluded to under the next suggested method of production of rheumatic fever by the streptococcus.

The allergic hypothesis was suggested by Swift (1925) and later by Zinsser (1928). This hypothesis is based on the fact that there is considerable similarity between rheumatic fever and tuberculosis, the symptoms of the latter disease being known to be allergic in origin. Further, by skin tests with various streptococcus products, rheumatic fever patients were shown to be sensitive, in that they have a twenty-four-hour type of skin reaction. Gibson and Thompson (1933) offered the first adequately controlled skin test study. These workers performed skin tests on large rheumatic and nonrheumatic groups. Similar ages, identical living conditions and opportunity of exposure to streptococcus infections were present in the two groups. There was little or no difference in the percentage of positive skin tests. In control groups which we have been able to study there has been relatively little difference in the percentage of positive skin tests of control children as compared with rheumatic fever subjects. There are many other objections to the allergic hypothesis. The experimental work supporting this allergic hypothesis and the clinical work indicate that man and animals, especially the rabbit, may readily become sensitive to the products of streptococci. No more definite connection has been established between allergy and rheumatic fever than the above.

It was recently suggested by Rinehart and coworkers (1934) that subclinical scurvy with additional streptococcus infection might well result in the human disease, rheumatic fever. Experimental lesions were produced in the heart of the guinea pig by deficiency diets and superimposed streptococcus infection. These lesions were not the characteristic lesions of rheumatic fever, but there were some points of similarity. In a series of guinea pigs we were able to reproduce these lesions with ease. However, the same lesions resulted in animals with acute scurvy and our work suggests that the lesions reported by Rinehart are the effect of complete scorbutus on the heart of the guinea pig. The skepticism of this etiologic mechanism is further warranted in view of a dietary experiment which we have performed at the House of the Good Samaritan, and the recent report of Sendroy and Schultz (1936). These observers present a detailed study concerning ascorbic acid metabolism and utilization in rheumatic fever subjects and controls. Their work does not support Rinehart's hypothesis.

The work of Coburn, of the Presbyterian Hospital, New York, is by far the most important recent work in connection with the etiology of rheumatic fever. The first report of this work appeared in a book called "The Factor of Infection in the Rheumatic State," which appeared in 1931. Many details and arguments must be omitted because of lack of time. Coburn is impressed with the association between hemolytic streptococcus pharyngitis and tonsillitis and subsequent rheumatic fever. He transported a group of eleven rheumatic fever subjects to Porto Rico and noted great clinical improvement in all of the patients. There was also a disappearance of hemolytic streptococci from the throats of these patients. Upon return to New York, hemolytic streptococcus pharyngitis was the usual precipitant of further rheumatic fever recurrences. Coburn presents further data concerning the geographic distribution of rheumatic and of scarlet fever and other hemolytic streptococcus diseases and believes this to support strongly the importance of the hemolytic streptococcus in initiating rheumatic fever. Far more important than the above are the immunologic data, most of which have been presented since the original report. Some data have been presented concerning agglutination tests, complement fixing antibodies and precipitins. However, the materials used for these tests were nonspecific antigens and, since his early reports, Coburn has paid but little attention to them. The more important immunologic test is the determination of antistreptolysin. This antibody was described by Todd (1932) who found by animal work that the hemolytic streptococcus is capable of producing a specific hemolysin. By immunization a specific antihemolysin developed, which has been called antistreptolysin. This antibody can be easily titrated and the technic in performing such tests is satisfactory and dependable. The association between hemolytic streptococcus infection and recurrent rheumatic fever has been previously noted. Coburn presents evidence that, in his series, the development of antistreptolysin occurs at the time of the recurrence of rheumatic fever. This immune body may be found in any individual with a recent hemolytic streptococcus infection. As a rule it appears at the end of the second or during the third week of the infection. This is the usual interval between respiratory infection and the symptoms of a rheumatic fever recurrence. Because of the above facts and others which I am unable to relate, Coburn feels that "rheumatic disease is a result of the following sequence of events: (1) Infection with toxin-producing strains of hemolytic streptococcus initiates a process peculiar to rheumatic subjects; (2) In the course of this process a substance is released, presumably from the antibody

producing tissues, which either directly or indirectly alters mesodermal structures. This substance is probably not the infecting organism, and at the present time there is no evidence to suggest that it is viable; (3) The release of this toxic substance seems to take place only when there is an immune response to hemolytic streptococcus." He further states that the evidence which he has collected indicates that the activity of the rheumatic process depends not only upon the effectiveness of the infecting strain of hemolytic streptococcus, but also upon the intensity of the immune response of the rheumatic subject to this bacterial agent.

This conception of the important rôle of the hemolytic streptococcus in rheumatic fever warrants serious consideration in view of the clinical significance of respiratory infection in rheumatic fever. It has been our experience that the association is a very close one. We have also observed that other agents or events seem to influence or precipitate recurrences of rheumatic fever. This suggests that streptococcus activity during respiratory infection might be of secondary rather than primary importance. These other agents or events (Bland and Jones, 1935) consist of an occasional non-streptococcus infection or disease, operative procedures, unexplained fevers, trauma and typhoid paratyphoid vaccine.

In order to evaluate the possible rôle of the hemolytic streptococcus in rheumatic fever, it was thought necessary to study a large group of control subjects from an immunologic standpoint. Such subjects were studied in a manner similar to that of the rheumatic fever subjects with respect to frequency of tests, etc. It has been necessary to collect data on a series of individuals from a wide variety of sources. They were collected with consideration of age, social strata, environment, disease and so forth. Data were obtained on 784 rheumatic fever subjects and nearly twice as many controls. The total series represents 2128 subjects and 4452 samples of serum. In the rheumatic fever group, recurrences of rheumatic fever followed 54 per cent of 255 observed sore throats. There were rheumatic fever recurrences following 32 per cent of 323 observed colds. Rheumatic fever recurrences resulted from 45 per cent of eighty-six various events considered to be of nonstreptococcal origin, these being 9 per cent of all recurrences. One hundred and twenty-three spontaneous recurrences of rheumatic fever without evident preceding events were observed, representing 30 per cent of the series of observed recurrences. It is hence evident that nearly 40 per cent of our observed rheumatic fever recurrences were not preceded by evident clinical respiratory infection. This was a higher percentage than was anticipated prior to analysis. There were severe recurrences of rheumatic fever in all of the groups regardless of the precipitating agent.

I will present a very brief analysis of our immunologic data, with especial reference to responses by antistreptolysin, which is easily the most reliable hemolytic streptococcus immune body, and by other immune bodies (antistreptolysin, antifibrinolysin and precipitins). It was found that of ninety scarlet fever subjects nearly 100 per cent had some hemolytic streptococcus immune body response. In fifty-six nonrheumatic children with severe sore throats, nearly 90 per cent developed immune bodies. In the rheumatic fever group, immune bodies developed in about two-thirds of the patients who had events, regardless of whether these events precipitated recurrences of rheumatic fever or not. In analyzing the data on the immune body response with regard to the event precipitating recurrences of rheumatic fever, it was found that a very high percentage of immune bodies developed as a result of sore throats, regardless of whether or not recurrent rheumatic fever resulted. The larger number of individuals with an immune response after colds did develop recurrent rheumatic fever. This was not true of the sore throat group, the percentages being practically identical regardless of whether or not the patient developed a rheumatic fever recurrence. Of the events considered to be nonstreptococcal in origin, about one-third of the patients developed an immune response whether or not there was recurrent rheumatic fever. This is in striking contrast to the groups following definite streptococcus infection in which an immune response occurred in over two-thirds. In the spontaneous recurrences of rheumatic fever, an immune response occurred in about one-third of the instances. These latter data show that in some of our recurrences of rheumatic fever, spontaneous and precipitated by seemingly nonstreptococcus events, there was activity of the hemolytic streptococcus, but that the development of an immune response in a small percentage in no way accounts for the fact that two-thirds developed definite clinical rheumatic fever without immune bodies.

In summary, it may be said that respiratory infection with secondary hemolytic streptococcus infection is a common precipitating agent in rheumatic fever. Should such an infection occur, the immune bodies will be found to develop in a patient regardless of whether or not a recurrence of rheumatic fever develops. Further, definite clinical rheumatic fever occurs without the development of an immune response, as shown by the failure of two-thirds of the groups with spontaneous recurrences and those precipitated by streptococcus events to develop hemolytic streptococcus immune bodies. Since this is true, it would seem that infection with the hemolytic streptococcus and the development of the immunological response to such infection will not explain all of the facts as we see them. There must, hence, be some further etiologic consideration involved.

That rheumatic fever may be caused by a filtrable virus has long been a conjecture. For about three years we have been doing occasional experiments in an attempt to determine a possible virus cause of rheumatic fever. These experiments have been carried out with a variety of materials, usually that obtained at postmortem or at an occasional subcutaneous nodule biopsy. The material has been treated in various ways and injected, chiefly into monkeys, by a variety of methods. Soon after beginning the work, one animal developed definite severe joint symptoms. He was not killed for two or three months, and postmortem findings were disappointing. About a year and a half ago a monkey developed severe joint changes and had typical nephritic edema about his eyes. He was killed on the eighteenth day and showed serofibrinous pericarditis, some suggestive microscopic changes in his heart and also subacute nephritis. This seemed to us to warrant extensive further study of this possible etiologic association. Further work has been disappointing. In May, 1935, there appeared a report from Schlesinger, Signy and Amies of England to the effect that they had obtained small bodies, by high speed centrifugation, from the pericardial exudates of rheumatic fever subjects. These bodies were visible by dark field examination and were not found in nonrheumatic exudates. Further, these workers reported these bodies as being specifically agglutinated by rheumatic fever sera. It was suggested that these bodies were similar to the so-called elementary bodies found in such well-known virus diseases as vaccinia, psittacosis and so forth. We have observed such bodies but have had great difficulty in agglutinating them. Upon succeeding

finally, they were found to be agglutinated by sera from normal individuals as well as from rheumatic fever subjects. There further occurred spontaneous agglutination in the antigen controls and small bodies indistinguishable by dark field examination were found in blood serum from a variety of sources. It is hence not clear whether the work of these observers will be reproducible and significant with regard to the etiology of rheumatic fever. If, however, this work proves to be of value, we must still produce the disease in some animal, and until this is accomplished I feel that the etiology of rheumatic fever will remain questionable.

DR. MORSE: I have listened to what Dr. Jones had to say with a great deal of interest but I still do not know what is the cause of rheumatic fever. Dr. White, will you tell us what the cause of rheumatism is?

DR. PAUL D. WHITE: I will beg the question by asking one of Dr. Jones, if I may. That is with reference to the attacks of acute rheumatism following various events. If I recall correctly, he said that 66 per cent of the attacks followed respiratory infections and 33 per cent followed nonrespiratory infection factors. I am wondering if that is about the ratio of occurrence of these happenings in the course of these patients' lives; in the winter, for example, are they more likely to have colds than to suffer from other conditions? Can he relate these percentages to the actual disturbances in childhood?

DR. JONES: First, I think Dr. Morse's remarks are important because we do not know the etiologic factors. With regard to Dr. White's question, 60 per cent of the observed recurrences of rheumatic fever followed respiratory events and in 40 per cent there seemed to be no respiratory infection factor. I agree that respiratory infection is common in rheumatic fever subjects and that one would expect such infection to be a common precipitating factor in this climate. Fully one-half of rheumatic fever subjects, certainly of those with recent active rheumatic fever, will show rheumatic fever symptoms after any respiratory event. It would seem probable that respiratory infection, as shown in our series, represents an important factor in the life cycle of the disease in this climate. The influence of the disease by such factors may be secondary and not necessarily of primary etiologic significance.

DR. MORSE: I think perhaps we can pass over etiology and go on to the pathology. I am going to ask Dr. Mallory to speak of the pathologic changes, which, as I understand it, are of two types, proliferative and exudative. I hope he will not only describe them but also explain to us why they occur. Now, being an old doctor and not up to modern ideas, I am especially interested in the Aschoff bodies, of which we hear so much, and I should like to know if the presence of any form of the Aschoff bodies means that the disease is still active. Do the Aschoff bodies ever disappear? Are the lesions of rheumatic fever ever healed? Of course the pathologist, when a child dies of rheumatism or rheumatic heart disease, always finds the lesions of rheumatism, and his point of view usually is they never get over it. But some of us practitioners, who clinically see children get well, grow up and have no trouble with their hearts, are rather skeptical, and I would like to know whether complete recovery is possible or not. As I said, I am an old-fashioned doctor, and I was brought up on chronic valvular disease and so on. Now they do not use that term, I think. But are not mistakes made by confusing the results of a previous pathologic process with active disease, for example, in the heart? That may be a foolish question for the modern man, but it is not for me.

DR. TRACY B. MALLORY: Ever since I have known Dr. Morse, he has been expecting the impossible of me. Now he wants me to tell you the pathology of rheumatic fever in ten minutes! I obviously must restrict myself to limited parts of the subject, and that means assuming that it is safe to leave out certain features. I am going to take it for granted that we are all starting with rheumatic fever in approximately the year 1920, and then I am going to try to give you a very brief survey of what has happened since.

About 1920, we all knew that rheumatic fever and chorea were associated with acute endocarditis, and that the serous cavities, particularly the pericardium, less often the pleura and peritoneum, were apt to show acute exudative phenomena. We knew that a great many hearts of people who died in rheumatic fever showed those peculiar things called Aschoff bodies. I think perhaps we can profitably start the discussion here by going into the subject of the Aschoff bodies, since a great deal of recent work has been devoted to them.

Various men in New York, particularly Louis Gross and Pappenheimer and von Glahn, men in Leipsic, Klinge, and his co-workers, have devoted the major part of their time in the last ten years to a study of the subject and have taught us many academically interesting facts. I am going to draw you a very rough diagram of the way in which an Aschoff body starts.

Throughout the body the initial attack of rheumatic fever, its virus, toxin or whatever the noxious agent is, seems to be upon the collagen fibers. If you look at a very early Aschoff body you will find a bundle of collagen fibres running along like that (drawing). The minute individual fibrils are cemented together into these rather homogeneous wavy bundles. Suddenly, at this point here let us say, the bundle swells up to two or three times the normal thickness and the specific staining qualities are more or less lost. Then at the far end of the lesion you can see the bundle emerging again and quickly becoming normal in character. If you stain this slide with silver stain for reticulum you can show that the fibers which make up the bundle have not disappeared in the center of the apparently necrotic area but run through it as individual fibers. When the fibers are fused together in the bundles they stain with aniline blue but fail to stain with silver, whereas as soon as they are separated in the lesion they stain readily with silver but not with aniline blue. So something evidently occurs to the binding substance of the bundle which permits the fibers to become separated, a hyaline material is deposited which renders the fibrils invisible with ordinary stains but they still run through the area relatively undisturbed, except in their staining reactions. Then around that area of altered collagen the peculiar large cells, about which pathologists and histologists have been arguing these last twenty years without settling anything, cluster. They evidently belong to the general group of mononuclear phagocytes, but more closely than that we do not know about them. Some of them appear to be produced *in situ* because we find mitoses in the immediate neighborhood; others without question emigrate there from a distant source. It is rather characteristic that they resemble to some degree the foreign body cells such as one sees in tuberculosis, but they never look quite like typical foreign body giant cells, and they often, as a matter of fact, look

surprisingly similar to the cells seen in Hodgkin's disease.

Exhaustive study in the last ten years seems to show that throughout the body this focal degeneration of collagen is the characteristic effect of the rheumatic virus. For years pathologists were primarily impressed by the characteristic little warty vegetations on the surface of the heart valves. But with microscopic study of the heart valves we have most of us come to agree with Ronald Grant that the vegetations are not particularly important and that the damaged connective tissues in the deeper part of the valve are probably the more important lesion. By intensive search for changes all over the body it has been shown quite clearly that rheumatic fever is a much more generalized disease than we supposed in 1920. Klinge in particular has done this with extraordinary diligence and with rather interesting techniques. He takes an aorta and twists it into a spiral so that the entire length of the aorta can be cut in single section. He does the same with other structures, such as the esophagus, and he has been able to show that nearly every aorta from a rheumatic case contains numerous Aschoff bodies, and that the entire esophagus, for instance, may be filled with them from the top to the bottom. He finds Aschoff bodies frequently in the trachea and in the capsules of the tonsils; they appear in subcutaneous tissues, of course, as rheumatic nodules. We find them in the capsules of joints. But I personally find it very hard to believe that, with the exception of the valvular lesions, an Aschoff body has the slightest functional significance.

The characteristic place to find an Aschoff body is well out in the adventitia of an artery. It is simply a localized little nodule that does not surround the vessel at all; it is in the outermost and least important layer of the vessel wall; and, although I think it is important evidence that rheumatism is present, I cannot believe that it has the slightest functional significance. On the other hand, in the heart valves the identical degeneration of the collagen, and the reaction to it, is what leads to permanent scarring and functional impairment.

It is customary to speak of acute myocarditis in active rheumatic cases, and we know that a great many patients show myocardial insufficiency with very slight valvular lesions in the acute stage of the disease. Personally, I think myocarditis is a misnomer. Clinically it means myocardial insufficiency. Pathologically it has been used to mean that Aschoff bodies are present but they bear no direct relationship in number or in size to the degree of heart failure that is present. You may have many Aschoff bodies and no heart failure. You may have severe myocardial insufficiency and to demonstrate a single Aschoff body is a major piece of research.

I believe myocarditis, that is, real degeneration of the muscle fibers, does occur in acute rheumatic hearts, but I have seen it only twice, and Dr. Jones, with wider experience in acute cases, has not seen it much more often.

So that it seems to me that myocarditis is not much more appropriate a term to apply to failing rheumatic hearts than it is a suitable term for describing an hypertrophied or dilated heart in hypertension.

We have discovered also in the last few years a lot of features about rheumatic fever which are interesting but, it seems to me, in practice not very important. We find that the large coronary arteries, the pulmonary artery, the aorta, as I said, are severely affected but the lesion is usually limited to the outer layers of the wall. Occasionally you get a moderate amount of dilatation and wrinkling of the wall but you don't get much scar formation, and I doubt if it is of any great importance. By a very intensive search we find that the peripheral arteries are quite often involved but the number involved in any one case, and in fact in a number of cases, is very slight. I do not believe the myocardial failure can be attributed to the few, usually minute lesions which can be demonstrated in the coronary arteries. One should compare these cases with periarteritis nodosa or with Buerger's disease before jumping to the conclusion that the lesions would be functionally significant. Vascular changes must in general be very diffuse or very numerous to cause symptoms.

These changes are particularly extensive in the lungs, and we have discovered another complication of rheumatic fever which is fairly common, the so-called rheumatic pneumonitis. There is very little evidence that it is a real pneumonia. From the clinical roentgenologic point of view these cases develop with astounding rapidity and disappear with even more astounding rapidity, sometimes two days later. Most of the cases that have been investigated have shown histologic extensive focal hemorrhages into the lungs and comparatively little else. Some of these cases show well-marked vascular lesions. Two cases which I, personally, have seen have not been combined with any demonstrable vascular lesion, although of course we have evidence of damage to small vessels in the form of hemorrhage, very likely analogous to the rheumatic purpura which you sometimes notice in the skin.

The question comes up finally, whether rheumatic fever causes damage to parenchymal tissues. It is common experience that the liver in acute rheumatism shows much more extensive degeneration than the degree of myocardial failure would justify, and most people are inclined to believe that this represents specific damage due to toxic injury by the rheumatic virus.

The other condition in which parenchymal cells have been definitely and consistently proved to be damaged occurs in the case of chorea. The number of cases of chorea which have been adequately studied pathologically is very small, but in those that I have seen reported degeneration of the ganglion cells has been very characteristic, along with slight perivascular inflammation such as you might find in any type of encephalitis.

To sum this up, it seems to me we have in the last ten years discovered a great many individual bits of information about rheumatic fever, but on the whole they tell us comparatively little about the production of the symptoms or the course of the disease.

CHAIRMAN HUNT: I should like to announce at this time the appointment of a Nominating Committee for the selection of officers for this Section for 1936-37, and I will appoint on that Committee Drs. L. W. Hill and E. W. Barron of Boston, and Dr. W. B. Adams of Springfield. For the benefit of those Fellows who have come in since the meeting opened, I would say that we would appreciate your sending your questions to the platform so that they may be answered and discussed by the panel.

DR. MORSE: Now that we have finished the pathology and etiology, we will really try to get down to business. I want to take up, in the first place, the symptomatology of rheumatism without apparent cardiac involvement, and I am going to ask Dr. Green to tell us what are the symptoms of an attack of rheumatism in a child when there is no apparent involvement of the heart.

DR. HYMAN GREEN, Boston: Rheumatic fever starts very insidiously. A child may have an acute tonsillitis, or upper respiratory infection, and

recover quickly. Then there will be a period of a week or so of perfect health. This period has been called the latent period. After this the following symptoms arise; namely, pallor, listlessness, lack of appetite, nose bleeds, joint pains, or abdominal pains. With this story, one may be sure that the child is developing rheumatic fever.

DR. MORSE: I will ask Dr. Stansfield whether he agrees with that.

DR. OLIVER H. STANSFIELD, Worcester: That sounds like a real case of rheumatic fever; but the cases that bother me more are the ones that turn up later with signs of heart involvement. For instance, within a year an orthopedic man treated a girl for fallen arches because she was complaining of her feet. Eighteen months later he found a very well-developed mitral stenosis. As to the relation of upper respiratory infection to rheumatic fever, I am glad to hear Dr. Jones say that he could not really connect the one with the other in many cases, because I never could. I do not know what the symptoms of rheumatic fever are in the atypical case, which is one of my chief worries.

DR. MORSE: What Dr. Green has said is very similar to what Dr. Kaiser said a while ago. Dr. Kaiser describes the manifestations of rheumatic infection, that is, a condition which is apparently continuous, as follows:—loss of appetite, pallor, chronic fatigue, fleeting pains, headaches, nosebleed, nervousness, and other vague complaints. Now these to me may be manifestations of rheumatic infection, but I wonder if there are not a good many other conditions in childhood which cause headaches, loss of appetite, pallor, nervousness, and other vague complaints. I am skeptical, personally, about saying that every child that shows these symptoms has a rheumatic infection. I think that there must be a lot of other causes for pains in children besides rheumatism. You get the ordinary social worker that is looking for cases, a series of cases of heart disease, for example, and she will find that every child has had a pain at some time or other. They all have pains at some time or other. I wonder if children do not have pains sometimes, not because they are growing, but because they have played hard and become tired, and whether they may not have some irritation of the epiphysis, and so on. And I should like to know what latent rheumatism really is. I do not like that term. I suppose a good many of us came here in automobiles, so that we all have the latent possibility of an accident before we get home. I should like to know what the "rheumatic state" is. That seems to me just a name. I can't see what it means unless it means an active rheumatic infection. Then why not call it rheumatism? I know I am trying Dr. Jones' temper in saying some of these things. I am going to ask Dr. Friedman to say a word or two first, and then we will give Dr. Jones a chance.

DR. FRIEDMAN: My conception of the rheumatic state is broad enough to include certain acute illnesses which are often associated together. They are polyarthritis, chorea, carditis, recurrent attacks of nose and throat infections, rheumatic nodules, growing pains and certain forms of rashes like erythema multiforme. All of the above conditions may be caused by rheumatic infection, and on account of the locality of the infection they have different manifestations.

When I see a child who shows any of the above-mentioned conditions, I consider the possibility of rheumatic infection being the underlying cause. If you have one symptom alone, the diagnosis may not be clear, but, when you have two or more symptoms associated together, the diagnosis of rheumatic infection becomes more apparent. We must, of course, rule out all other conditions with similar symptoms before we make the diagnosis of rheumatism. When our knowledge of the bacteriology of rheumatic infection is more clearly established, the diagnosis will become much more definite. When a child shows pallor, loss of weight and complains of so-called growing pains, one must bear in mind the possibility that this child may be suffering from rheumatic infection.

DR. MORSE: I will now ask Dr. Jones to say what he thinks about it.

DR. JONES: I agree heartily with Dr. Morse. A definite diagnosis of rheumatic fever can be made only when the clinical picture is more or less complete. I prefer the term "rheumatic fever" or "rheumatic fever subject" to that of "the rheumatic state". To make a diagnosis of latent rheumatism is difficult, and I should say impossible unless the child develops evidence of rheumatic heart disease or some manifestation of it, say a prolongation of auriculoventricular conduction by electrocardiogram. In this connection I believe the terms "rheumatic state" or "prerheumatic child" are clinically misleading, and will remain so until we have a definite test for the disease, rheumatic fever. Hence, if there is vague symptomatology in a child without a previous definite history of rheumatic fever, the development of rheumatic heart disease would seem the only definite proof of the exact nature of the illness.

DR. MORSE: Dr. Green wants to talk about pains.

DR. GREEN: I think the question of pains should be investigated thoroughly because the history of pains may be misleading. I recently had a case of a boy of eleven years who complained of pains all over, making it difficult to tell from the story whether he had rheumatic fever. He had flat feet and poor body mechanics. After giving him plates for his feet and exercises for his posture, he was able to walk well and did not complain of any more pain. Therefore flat feet and faulty posture should be considered in every history suggesting rheumatic fever.

DR. MORSE: I am interested to hear what Dr. White has to say about this.

DR. WHITE: In our consideration of the symptoms, if we exclude other conditions after a child has been ill for a few weeks, and other tests, such as the electrocardiographic examination, of which I will speak a bit later, indicate some damage to the heart, or if there is evidence of enlargement of the heart, we may make a diagnosis of rheumatism even without rheumatic pains. If my own experience is correct, definite rheumatic pains and arthritis are much more common in older children and in adults than in the youngest children at about the age of four or five years. I would like to ask Dr. Jones if that is his experience too.

DR. JONES: I think that the older the child, the more apt he is to have definite joint involvement; but we do see it at times in the very young. It is, however, not an important feature of the disease syndrome. In our series of some 1400 rheumatic fever subjects about 70 per cent had varying degrees of joint symptomatology at some time or another.

DR. MORSE: I suppose everybody agrees that rheumatic nodules are a positive proof of rheumatism. I will ask Dr. Green his opinion.

DR. GREEN: As far as rheumatic nodules are concerned, it takes keen observation and considerable experience to find them. Most men in practice do not know what they are and have never seen them. It is important to search thoroughly for them, as they are pathognomonic of rheumatic fever. They frequently occur on the top of the head and may be the size of a marble. One was recently removed in a large Boston hospital for a question of sarcoma but on microscopic examination it proved to be rheumatic fever.

DR. MORSE: I would like to hear from Dr. Jones.

DR. JONES: This would seem to be a question for Dr. Mallory. I have rarely seen erythema nodosum in our rheumatic fever subjects. A few years ago Dr. Sprague reviewed all of the erythema nodosum series at the Massachusetts General Hospital, and found that in only a few instances did any of the patients have rheumatic heart disease. The work of Collis is pertinent. This English observer demonstrated that the lesions of erythema nodosum could be reproduced in about one-half of his patients by the injection of tuberculin. In the other half the lesions were reproduced by streptococcal products. There seems very little evidence of any close clinical association between erythema nodosum and rheumatic fever.

DR. MORSE: Dr. Mallory.

DR. MALLORY: I was not sure from what Dr. Green said whether he thought there might be accidental confusion between erythema nodosum and rheumatic nodules. From the histological point of view the two are entirely distinct, that is, one can easily recognize the rheumatic nodule.

DR. MORSE: I want to take up next the symptomatology of cardiac involvement in rheumatism. All agree that involvement of the heart is common, and that it is the one serious thing in rheumatism in early life. All agree that all parts of the heart are involved, that is, that it is a carditis. How common is involvement of the heart? Is it always involved in every case, even when there are no symptoms? I suppose it always is in fatal cases. Is it in cases in which no signs of involvement are apparent? What are the symptoms and signs of involvement of the heart? How shall we make the diagnosis? Some of the points which I think ought to be considered are the temperature, the rate of the pulse, the size of the heart. Now when we come to the question of the size of the heart, can we determine it by percussion or not? If we do, do we go by the absolute size of the heart as we determine it, or by the relation of the diameter of the heart to the diameter of the chest? Do we have to take x-rays of the heart in order to determine the size? What is the comparative importance of the size as determined by percussion and by x-ray? Then murmurs, and here we have to take up the differentiation of functional and organic murmurs; the white count and Schilling's differential count; the electrocardiogram; the sedimentation test. Now those are all methods to be used. How about the general practitioner who cannot do the sedimentation test, probably cannot get an electrocardiogram, or even an x-ray. He can do a white count. He can percuss. How well can he get on with his ears, hands and brains alone without the sedimentation test, electrocardiogram, and so forth? Then a little later I want to find out what "potential" heart disease is. Still later I want to find out how to diagnose a new infection of the heart in a fresh attack of rheumatism in a child who has previously had involvement of the heart. I hope they can tell me. Dr. Friedman, suppose you tell us something about the symptoms and signs of involvement of the heart.

DR. FRIEDMAN: As I said before, when the involvement of the heart is not marked, it is much more difficult to make the diagnosis. When you have marked involvement with definite hypertrophy the diagnosis is obvious. When the child has fever, signs of dyspnea and precordial pain, the possibility of carditis should be suspected even though careful examination does not show any murmurs. Once again I want to stress the importance of erythematous rashes which are so common in rheumatic infections and so often overlooked as insignificant.

If you suspect rheumatic infection and your attention is focused on the heart action, you will often find evidence to corroborate your suspicions. The mother may volunteer the information that the child tires easily, does not join other children at play and gets out of breath on slight exertion. The milder the infection and the milder the symptoms, the more difficult the diagnosis will be. If, however, you keep the possibility of rheumatic infection in mind and remember that it may have various manifestations, the diagnosis will be less difficult.

DR. MORSE: I think perhaps before we take up the comparative value of the symptoms I will ask Dr. White to say a word or two about the electro cardiogram in connection with the heart in childhood.

DR. WHITE: Before I begin the discussion on electrocardiography may I ask two important questions of Dr. Mallory? One is with reference to the significance in later life of coronary involvement by rheumatism in youth, that is, the question of serious coronary disease induced by a previous rheumatic infection. It is my own belief that rheumatic coronary involvement is not an important factor in later life. And the other question relates to the belief of Coburn and others that there may be extensive early changes in the myocardium, hemorrhagic or otherwise, before the Aschoff bodies are formed, that is, that the Aschoff bodies are rather late manifestations, and that serious lesions may occur prior to their appearance. I would like to have Dr. Mallory answer those two questions.

DR. MALLORY: In answer to the first question I can only say that from the material which has fallen within my experience I have not been impressed with the importance of coronary artery involvement in rheumatic fever. It is perfectly true that children dying with acute rheumatic fever will often show early acute atheromatous lesions in the coronaries; but Dr. Timothy Leary and others have shown that acute lesions of the coronaries follow a great variety of acute infections in childhood. It is probably rather rare but I do not doubt it happens in rheumatic fever. I question whether it happens oftener, however, than in any other acute infection.

As to the second question, as I said before, I have seen two cases with very acute myocardial degeneration in acute rheumatism. The experience everywhere, however, is that it is a comparatively rare thing. In twenty acute rheumatics dying in the acute stage of the disease, I doubt if more than two or three would show degeneration. If Coburn has more than that, I think he probably has had a rather exceptional group of cases. However, it is a type of thing that will have to be checked in a very large number of cases before we are sure, and no one person sees enough acute rheumatic cases to get very satisfactory statistics.

DR. WHITE: I believe that almost everyone will agree that the heart is practically always involved

in the acute rheumatic infection, but that the involvement may be so slight in some cases that we never have any signs or symptoms from it. Probably about 20 per cent of the rheumatic cases and a very large percentage of the cases of so-called pure chorea escape evident heart damage.

It has been shown that the electrocardiogram has an important use in cases of myocardial involvement of any sort. If there happens to be a lesion in the bundle of His or other conducting tissues of the heart, if the myocardium itself is extensively involved, or if the pericardium is seriously affected, we will find changes in the electrocardiogram, which may thus be a useful record in the study of the rheumatic infection.

My own interest in the subject dates back to twenty years ago when we discovered, in the Outpatient Department of the Massachusetts General Hospital, a young man, aged eighteen, who showed by graphic record partial heart block (at first clinically diagnosed as sinus arrhythmia). It was more or less by chance that we took the graphic record. This heart block developed a few days after tonsillitis and just preceding an attack of acute rheumatic fever and so was the first conclusive evidence of rheumatic infection. The heart block lasted only a few days and ten years later I found the heart to be perfectly normal.

Since that time our experience with the electrocardiograms of some hundreds of cases of acute rheumatism in childhood has demonstrated, as has that of others, a good many instances of abnormalities which are usually corroborative of other clinical evidence of the infection but occasionally are important signs in themselves of the presence or the existence of active infection; rarely do the abnormalities become set or chronic. Although these abnormalities are not pathognomonic of this infection, they are found in it more often than in any other infection in childhood, barring severe uncontrolled diphtheria.

A review which I made yesterday of fifty recent cases of acute and subacute rheumatism at the House of the Good Samaritan in Boston will well illustrate what we may expect to find. From one to fifteen electrocardiograms daily to monthly were taken in each of these cases, averaging five or six apiece. There were fifteen males and thirty-five females, ranging in age from two to twenty-two years, with the great majority (forty-four) ranging from five to fifteen years, inclusive. Nine of these fifty cases died. Autopsies in seven confirmed the diagnosis; permission for an autopsy was not obtained in the other two cases.

The electrocardiograms were wholly normal in thirty-six of the fifty cases, or 72 per cent, including all eight cases of uncomplicated chorea and all six cases of chorea complicating rheumatic fever, and also including four of the nine fatal cases.

Heart block of the auriculoventricular type, partial in degree with prolonged P-R intervals (up to 0.25 second) and without dropped beats, except in one case with 2: 1 block, was found transiently in eight cases (16 per cent), including three of the nine fatal cases. In one of these eight cases digitalis was quite probably responsible, at least, in part.

Bundle branch, or intraventricular, block was found in slight degree in but one case with left axis deviation.

Sinoauricular block (unimportant) was present in one.

Auricular premature beats were found in one case and ventricular premature beats in none.

Auricular fibrillation occurred in one case, a girl of nineteen years with mitral stenosis and a history of a previous attack of rheumatic fever five years before.

Left axis deviation of abnormal degree was found in two cases, one of whom had aortic regurgitation. The R wave in lead 2 was split in one other case. There was no case of abnormal right axis deviation.

The T waves were abnormal in six cases, very high in one, inverted or diphasic in Leads 1 or 2 in four, two of which were digitalized, and inverted, of coronary type (probably due to pericarditis), in one case.

The following additional statistics may be of interest:

Massachusetts General Hospital Electrocardiographic Series	Bundle branch block 328 cases, with a history of rheumatic fever in 47 (14 per cent).
	Complete auriculo-ventricular heart block 72 cases, with rheumatic heart disease in 3 (4 per cent).
	Right axis deviation 288 cases: 53 per cent had mitral stenosis.
	Mitral stenosis 100 cases: 51 per cent had abnormal right axis deviation.
	Aortic regurgitation 50 cases: 40 per cent had abnormal left axis deviation.
	Big P waves 132 cases: 72 (55 per cent) had mitral stenosis.

King's series of 150 cases of bundle branch block contained fifteen rheumatic cases (10 per cent).

There have been even a very few cases of the Adams-Stokes syndrome in rheumatic fever like Faulkner's case of 1930.

Conclusions: Electrocardiograms in rheumatic fever are useful but not often essential. They may be normal in fatal cases and abnormal in mild cases but the more severe the infection the more likely are abnormalities to be found. In that respect the electrocardiogram in the rheumatic infection is somewhat comparable with the electrocardiogram in coronary disease.

DR. MORSE: I see many cases from time to time in which physicians have mistaken murmurs made outside the heart, or functional murmurs, for organic murmurs. It is very important to distinguish between them when you are making a diagnosis of rheumatic heart disease, and I will ask Dr. Stansfield to say a word about the differential diagnosis of these conditions, remembering that we are talking about acute rheumatic heart disease, not about chronic.

DR. STANSFIELD: This looks to me like a good place to display ignorance. I suppose we all do agree that functional murmurs exist, at least we hear a soft murmur which is not transmitted in any direction and does not seem to mask any of the normal heart sounds. We will assume the first time you see the child in perfect health he may display a murmur and finally conclude because it is not being propagated in any direction that it must be a functional murmur, whatever a functional murmur is. But if the child is sick, as Dr. Green described, when these children are in more or less chronic ill health, it is a little bit more of a problem to decide whether you are dealing with what I will call a nonpathologic murmur or a pathologic murmur. Of course you go by the signs that you find around the heart.

I think by taking the general status of the case,

particularly the manifestations of abnormality about the heart and other symptoms, you can come to a reasonably just conclusion as to whether you are dealing with a functional or organic murmur.

DR. MORSE: Dr. Stansfield has already answered the question I was going to ask the others, that is, the question of percussing out the size of the heart. I am going to ask each one in turn. What do you say, Dr. Friedman? Can you percuss the size of the heart?

DR. FRIEDMAN: It all depends on whether the hypertrophy is slight. In marked hypertrophy it is very easy to demonstrate it by percussion. When the hypertrophy is slight, I rely more on the position of the apex beat and I like to check my findings with an x-ray plate.

DR. JONES: Unless you are dealing with extremely thick chests, or patients with large breasts, I believe it is possible by percussion to determine the size of the heart fairly accurately.

DR. GREEN: I think it is possible to determine the size of the heart particularly in older children. In the hospital the x-ray is used as a further check on percussion. The greatest difficulty is to determine the heart size in infants.

DR. MORSE: The reason you can't percuss the size of the heart of an infant is because you hit it too hard.

DR. WHITE: Most children have chest walls that are thin enough to allow fairly accurate percussion of the heart if the doctor is willing to take time enough to learn how to percuss, but so many physicians and medical students in recent years have been taught that they must have an x-ray examination of the heart that they have lost the art of percussion or never acquired it. I am quite certain that cardiac percussion should be actively taught again. It is more difficult in adults to be sure, but in children, as a rule, the heart can be reasonably well percussed. One should always percuss in the third left interspace as well as at the cardiac apex because if there is marked prominence of the pulmonary artery one can almost always determine it by percussion.

DR. MALLORY: I naturally would not be rash enough to try to percuss a heart but I probably have had more experience than any person present in checking other people's percussions, and I emphatically say there are some people who can percuss a heart fairly accurately in a good many chests; there are some chests in which nobody could possibly percuss a heart; and there are some people who can't percuss a heart correctly in any chest.

DR. MORSE: We are getting a little bit behind time, I am afraid. I think we can all tell what the temperature is and can also count the pulse all right, and apparently, if you know how, you can percuss out the size of a heart, and, if you use your ears, you can tell the difference between functional and organic murmurs. It is, of course, not difficult to make a white count. Practically all children that are sick have an elevation of the white count, whether they have cardiac involvement or not; so that, to my mind, it is not of a great deal of importance. Those who do not agree with me will hold up their hands. (Nobody held up his hand.) Schilling's differential count is of some importance. Dr. White has told us about the electrocardiogram. Nobody has said anything about the sedimentation test. If any of you have ever seen it done, you know it takes a long time. I will ask Dr. Jones to say what it is in about a minute.

DR. JONES: The sedimentation rate is certainly of distinct value. It is probably of more value clinically than the white blood count, but is not an absolute criterion of active rheumatic fever. However, if the test is performed by the method of Ernstene and Rourke, in which there is a correction for anemia, one learns not only an indication of active infection, but also whether the patient needs iron therapy.

DR. MORSE: Now I want to ask you all the same question, the one I referred to in the beginning. That is, can the general practitioner make the diagnosis of cardiac involvement in rheumatism in childhood with his ears, hands and brains alone? Yes or no. Dr. Friedman.

DR. FRIEDMAN: Yes, on the whole, but I qualify it.

DR. JONES: Yes.

DR. GREEN: Yes.

DR. STANSFIELD: Of course you can.

DR. WHITE: But we must distinguish between temporary and permanent involvement of the heart. We have difficulty in doing this often at first; we sometimes must wait to see whether the signs disappear before diagnosing permanent valvular disease.

DR. MORSE: Here is a question from the floor for Dr. White to answer.

DR. WHITE: Are there electrocardiographic changes in young children with only transient pains? Rarely. The electrocardiogram in a study of rheumatic cases is somewhat like the electrocardiogram in the study of coronary disease. We may have severe coronary disease with angina pectoris with a normal electrocardiogram. That may also happen with rheumatism. We may have fatal cases with normal electrocardiograms, or we may have in a mild case a change in the electrocardiogram, but as a rule the more severe the infection the more changes in the electrocardiogram.

DR. MORSE: Now we come to treatment. Everybody agrees, I suppose, that the child with acute rheumatism, whether it has or has not involvement of the heart, should be put to bed and kept in bed. Now the question is how long should a child with acute rheumatism, without apparent involvement of the heart, be kept in bed? How long should one be kept in bed with involvement of the heart? How are we going to decide rightly? How is the general practitioner going to decide? I think I will ask each one to state briefly what his opinion is on that subject.

DR. GREEN: The most difficult question of all is how long to keep the child in bed. In the past few years, the sedimentation rate has proved a valuable laboratory procedure for the presence or absence of infection. When it is normal, it is good evidence that the infection has subsided. Heretofore we have relied on our clinical evidence, namely normal temperature and normal pulse rate. In the home, we should allow a child up when the color has improved, the appetite has returned, when there is a gain in weight, and normal temperature and pulse.

DR. JONES: I firmly believe that rheumatic fever subjects should be kept in bed much longer than their clinical manifestations last. Further, I believe that laboratory studies are of great value in deter-

mining the presence or absence of active rheumatic fever, and as a therapeutic guide. Clinically, evident heart disease may develop when there is only laboratory evidence of infection. I also feel that rest is important so long as there is any evidence of infection. Without laboratory studies, the general practitioner will necessarily have to be very cautious in evaluating the clinical picture and allowing the patient to resume physical activity. Laboratory evidence of infection may persist for long periods, and at this time there is a peculiar susceptibility to recurrences of rheumatic fever. There is good reason, therefore, to insist on long bed rest.

DR. FRIEDMAN: You can't generalize on this subject. If the general condition of the child is improving, and the infection as shown by the temperature and pulse is quieting down, we can think about letting the child up, but even then it is better to err on the safe side by keeping him in bed a while longer.

DR. STANSFIELD: I have always had the feeling that rheumatic fever was somewhat analogous to tuberculosis, and that the treatment should be along that line. My own habit is to go very slowly and feel skeptical about recovery to the extent of not letting patients up in less than three months. Of course it does happen that they may get up sometimes sooner than that, but if you permit that you want absolute evidence, all the evidence you can get, that it is safe to do it, and if there is any doubt about it a little bit longer won't hurt.

DR. MORSE: I have a question here for Dr. White. How long in weeks or months approximately?

DR. WHITE: It is not only because of the infection that we need to treat these patients with prolonged bed rest; involvement of the heart is equally important in demanding rest. The length of time will vary from weeks to months, even to years, as the individual cases require. It used to be the custom in the treatment of coronary thrombosis to allow the patient to be up after a few days, with the risk of a permanently dilated heart. It is quite likely that the heart in acute rheumatic fever will dilate more or remain more dilated and damaged if the patient is allowed to be active too early. This seems probable but we have little or no proof. We are trying to collect that proof now. The difficulty lies in securing enough control cases.

DR. MORSE: There are one or two questions as to drug treatment. In the first place, how about salicylates? Do you use them or not? What do they do? What is the dose?

DR. FRIEDMAN: Yes, I use them because they relieve the symptoms and make the patient more comfortable. Whether they have any specific action on the infection I do not know.

DR. JONES: I do not believe that salicylates are specific. We use them frequently for the comfort of the patient, but do not expect them to prevent the development of or increase in heart disease. There are two cautions in the use of salicylates which seem pertinent. When patients are over the acute stage of rheumatic fever it is unwise to keep them on large salicylate dosage, because the symptoms are masked, and intelligent therapy would necessitate knowing whether the patient had mild clinical symptoms. Salicylates hence give a false sense of security. Neither is there any real evidence that salicylates prevent the recurrence of rheumatic fever. The prophylactic use of salicylate was some years ago advocated by English observers. In reviewing their series, the first record reviewed was that of a child with mitral disease. Following a severe sore throat massive doses of salicylates were given for two or three months. Though clinically well during the time, aortic disease was found when the child was re-examined. This indicates to me that rheumatic fever had been active and the symptoms masked by salicylates.

DR. GREEN: Salicylates are used at first up to sixty or more grains a day, reducing the dose according to the relief of pain. We have used salicylates during the period when the child is up and about with the idea of attempting to prevent recurrences. This procedure has been carried on in our clinic for about ten years but as yet we have not collected our statistics to prove this point. Our feeling, however, is that it has made some difference in the number of recurrences.

DR. STANSFIELD: I use salicylates for the same reason that the other gentlemen have given, although I sometimes wonder when I order them how much tradition there is in the business.

DR. WHITE: During the World War there was an epidemic of rheumatic fever in a certain camp in France, and a considerable number of patients came into our hospital from that camp within a comparatively short time. These patients were separated into two groups; to one group we gave large doses of sodium salicylate with sodium bicarbonate, while to the others we gave phenacetin and other analgesic remedies. The group that took the salicylate quickly cleared up. The joint symptoms disappeared within twenty-four to thirty-six hours, and the fever went down rapidly. The patients in the other group after two or three days were still very uncomfortable. They learned of the experiment, and we could not continue it any longer. There was not the least doubt about the specificity of the salicylates so far as the arthritis itself was concerned. On the other hand in young children with acute rheumatic heart disease and very little or no arthritis, it is unwise to use salicylates regularly because they are likely to mask evidences of active infection.

I should like to ask Dr. Morse a question, if it is permissible. Does he think that salicylate therapy may control a pericardial effusion?

DR. MORSE: I am afraid my experience is rather limited, but I should say no.

The next question I want to ask is, is it proper to give digitalis for rheumatic heart disease when there are no signs of cardiac failure?

DR. WHITE: No.

DR. STANSFIELD: No.

DR. GREEN: No.

DR. JONES: No.

DR. FRIEDMAN: No.

DR. MORSE: There is a question here just handed up, whether digitalis should be used when there is acute rheumatic fever with evidence of cardiac decompensation. I should answer yes, though it probably would not do much good if the involvement of the heart is acute and there is no chronic lesion. I will let the others say what they think.

DR. WHITE: I think it helps but I am not certain about it. It is always worth while to try it in adequate dosage but during a very active infection it may be valueless.

DR. STANSFIELD: I have nothing to say.

DR. GREEN: I think it should be given in acute decompensation.

DR. JONES: I have occasionally seen good results from it but have often wondered whether it was due entirely to the diuretic effect.

DR. FRIEDMAN: When it can do no harm and may do some good, I should use it.

DR. MORSE: The questions are coming up here thick and fast. The next question is: Do you prefer sodium salicylate to aspirin? Dr. Jones.

DR. JONES: We use aspirin almost entirely, and because it is better tolerated by the average child than sodium salicylate.

DR. MORSE: Now a word or two as to preventive treatment. When the acute attack is over, with no apparent cardiac involvement, or cardiac involvement but no symptoms, what do you recommend? I am afraid there is not time for everybody to say what he does about that. Perhaps we will ask Dr. Green to tell us in two minutes what he does.

DR. GREEN: In the way of preventive treatment, foci of infection such as bad teeth, and infected tonsils, should be removed and efforts should be made to increase weight gain by proper nourishment and rest periods.

DR. MORSE: I think a good point to be remembered is that a few years ago these children were too much limited in their exercise and in their lives, and that probably it is safe to let them do more than they have done in the past, that they are better for it. I wonder if the gentlemen here agree? [Answer in the affirmative.]

The next question we have is as to the use of salicylates in preventive treatment. I think that has been pretty well answered already, and I should think that the gentlemen here would not agree with Kaiser's continuous treatment with aspirin and magnesium oxide.

The next thing I have here to discuss is the tonsils, whether to remove them or not, and the results to be expected. If we had another session this afternoon we might be able to discuss this matter in detail. My general impression is, based on the work of Kaiser, that removal of the tonsils possibly removes a certain predisposition to rheumatism, but that if the tonsils are removed after the child has had rheumatism and rheumatic heart disease, it does not diminish the number of attacks.

There is a question here as to how much attention should be paid to the sedimentation test in deciding when to remove the tonsils. That of course will bring up the question also as to whether the tonsils should be removed in an acute attack, or whether one should wait until the acute attack is over. I think perhaps I will ask Dr. Jones to answer that question, and if the others disagree they can say so.

DR. JONES: I have not personally had any experience with the removal of tonsils during acute rheumatic fever. At the Boston City Hospital, whence the suggestion came, I understand that the procedure is rarely carried out at present. There has been increasing skepticism during the past few years of the value of tonsillectomy. It further appears evident, that, if tonsillectomy is determined upon, the operation should be performed when there is no evidence of rheumatic fever, clinical or laboratory. Many recurrences of rheumatic fever could be avoided if this caution were generally applied. I have seen fatal issues result from recurrences apparently precipitated by tonsillectomy. Since the beneficial result of such an operation is questionable, it would seem wise to err on the side of safety and remove the tonsils only after the disease has subsided.

DR. GREEN: I should like to ask Dr. Mallory how often he has found Aschoff bodies in tonsils.

DR. MALLORY: I have never personally looked for them but Klinge finds them in large numbers in a large percentage of cases, in the capsule of the tonsil, that is, the connective tissue just behind the tonsil itself. I think there is no reason to question his figures.

DR. MORSE: Dr. Friedman, if all your cases were rich, would you send all the children with "latent", "potential" rheumatic heart disease South for the winter, and, if you would, why would you do it? Would it be because you believe in that kind of treatment or because of some other reason?

DR. FRIEDMAN: I would not in every case. I would keep most of them here and see that they are made free to gain by proper nutrition and hygiene. I feel that the fact that we do not see so much rheumatic infection in the well-to-do who can afford proper nutrition and hygiene, would indicate that as a whole we can handle the problem here without sending them South. If they felt that they ought to go South, I would tell them to go right ahead.

DR. JONES: I do not agree. Rheumatic fever patients spending the winter months in a subtropical climate do improve faster than those remaining in this climate. Transportation is, however, not a panacea. If a patient be so economically situated it would be wise for him to go to a more ideal climate, but this should be done during the winter and spring months for five or six years. Few people are able to do this.

DR. MORSE: There seem to be many questions about digitalis for Dr. White to answer.

DR. WHITE: May I add a word about sending children South? A few years ago, in Jamaica, in talking with a capable doctor who had practiced actively there for thirty years, I learned that he had never seen a case of rheumatic fever in a child, that he had not encountered chorea, and that he had never seen a case of valvular disease in a child, except in a few rare congenital heart cases. Coming from there into southern Florida, we conceived the idea of sending some children to Miami Beach, which we did for five successive years (1930-35). We found that, although these children were benefited, they were not apparently benefited so much as they might have been in a more tropical climate such as exists in Jamaica.

I am quite convinced that, if a family is riddled with rheumatic infection and rheumatic heart disease and that family can go to a tropical climate, it should be sent there because there is no doubt of the benefit of a climatic change.

As to the question about digitalis, it is asked whether there is danger from it in an acute rheumatic attack. I do not think there is any danger, but I do believe the drug is often ineffective. I have not seen any harm come from the use of digitalis in these children and I have often seen it of some value. Once in a while, when digitalis fails to control the heart rate in a young person with auricular fibrillation and active rheumatism, it is worth while to resort to quinidine, although that drug is not ordinarily indicated in serious heart disease and in fact is supposedly contraindicated. We have, however, saved at least one life, and I think one or two others, by its use in restoring normal rhythm.

As for the dosage of digitalis in children, it should be relatively somewhat larger than the adult dose. It may be figured out at approximately 2/10 of a gram for each ten pounds of body weight. However, one must individualize for each case.

DR. MORSE: An attempt has been made in New York to desensitize children to rheumatism by intravenous injections of streptococcus products. What do you think about it, Dr. Jones?

DR. JONES: It would seem unjustifiable in view of the report of Wilson. In her series, there was no appreciable variation in the number of recurrences in three groups: 1. Patients desensitized or immunized with the intravenous injection of the best vaccines available; 2. A control group receiving typhoid vaccine intravenously; 3. An untreated group. In addition, the patient is put through some very stirring episodes in the process of the treatments.

DR. MORSE: I have a very good question here from the floor: By what criteria does the clinician determine when an active rheumatic process has become inactive? Dr. Green.

DR. GREEN: That is a difficult question. Close observation of the patient as to temperature and pulse rate, general condition of the patient, and the response to bed rest are the chief criteria of improvement. Complications may arise early in the disease so that the quicker the diagnosis is made the better.

DR. JONES: I am sorry to do so much of the talking, but we have been impressed on occasion with the fact that active rheumatic fever may persist for long periods with but little evidence. This is demonstrated in the occasional patient coming to autopsy, in whom there has been little or no clinical or laboratory evidence of active rheumatic fever. We had one adolescent girl, who for some time had been comparatively well. During the afternoon of a very hot day she suddenly had a chill and high fever without apparent cause, and death resulted within several hours from pulmonary edema. Pathologically, she showed extensive active rheumatic fever. I also recall a second patient dying within twenty-four hours of uncontrollable auricular fibrillation. Active rheumatic fever was also found postmortem.

DR. MORSE: Just a word or two as to prognosis. Does recovery from rheumatism ever occur? That is, is susceptibility to infection ever overcome? Is the allergic condition ever overcome?

DR. WHITE: It is certainly true that many adults coming into middle age are completely free from recurrences of acute rheumatism to which they were subject when they were children; in other words, patients can make a good recovery from a tendency to rheumatic recurrence, even though acute attacks may rarely occur as late as sixty years of age.

DR. GREEN: I think there can be recovery from rheumatic fever particularly in children. We have had children in our heart clinic who have had attacks of rheumatic fever, in some cases with severe pericarditis, who after being followed for ten years now show absolutely no evidence of rheumatic fever.

DR. JONES: I agree. I do think that rheumatic fever can occur in a patient who may never have it again, and who remains perfectly well. I also think that when we know the etiology and have a definite test for the disease, we will find that a greater percentage of the population has rheumatic fever than is now evident.

DR. FRIEDMAN: I think they do definitely recover but I think the fact they have had it should make one watchful to see whether any recurrence or relapse can come later on.

DR. MORSE: The next question I have is: Are lesions in the heart ever healed? Dr. Mallory, I think, has practically answered that question. I can say for myself that I think they are. Recovery from rheumatic heart disease in childhood is entirely possible and does happen. If there is anybody who does not agree with me, speak up.

DR. WHITE: I do not disagree, but I wish to add a word with reference to the interpretation of murmurs, which very frequently have been wrongly judged. A systolic murmur at the apex is sometimes attributed to damage to the mitral valve when it is really due to the effect of left ventricular dilatation. Also, it is interesting to note that occasionally acute rheumatic changes in the heart are found, entirely unsuspected, on postmortem examination of individuals dying noncardiac deaths.

DR. MORSE: I have a question here: In those children with acute follicular tonsillitis and concomitant joint pains without swelling and of short duration—should we treat them as rheumatics? I will ask Dr. Stansfield to answer.

DR. STANSFIELD: I do not see why we should. Practically every acute tonsillitis is going to have pretty definite discomfort around the joints as well as the muscles, and there is no reason to treat it as rheumatic fever.

DR. MORSE: Do the rest agree? Apparently they do. Another question from the floor: How important is weight gain or loss in prognosis? We will let Dr. Friedman answer that.

DR. FRIEDMAN: I think it is very important because any gain in weight is direct evidence that the child is doing better and is overcoming the systemic infection. I think it is of very great importance in guiding us as to prognosis.

DR. MORSE: The time is getting short, and we shall not get through all the subjects which I had intended to have discussed. I think we shall have to stop at this point. I had hoped to take up chorea but, as it is now approaching half past eleven, when we must adjourn, I will turn the meeting over to the Chairman.

CHAIRMAN HUNT: We have one matter of business to attend to, namely, the election of officers for the ensuing year. The Nominating Committee presents the names of Dr. James Herbert Young of Boston as Chairman, and Dr. James M. Baty of Belmont as Secretary. Are there any other nominations from the floor? If not, what is your pleasure? It is moved and seconded that the Secretary cast one ballot for the slate as nominated. Those in favor will say Aye, those opposed No. It is a vote. The Secretary has cast his ballot as directed, and you have elected Dr. J. Herbert Young of Boston as Chairman of the Section for the ensuing year, and Dr. James M. Baty of Belmont as Secretary.

In behalf of the Section and expressing my own gratification, I feel that this has been an outstanding Section meeting of this Annual Meeting of the Society. I know that, without any question, those of us who have been at other meetings during the past three days have noticed more questions coming to the platform, and we have had much more discussion and more general interest from a great many men who would not get up on their feet, come to the microphone, and ask questions. And, on behalf of the Section and myself personally, I want to thank our galaxy of stars here—Dr. Morse, Dr. Friedman, Dr. Green, Dr. Jones, Dr. Mallory, Dr. Stansfield, and Dr. White—for their interesting discourses. I think they have given us an outstanding demonstration in rheumatism, which affects the practice of every one of us almost daily. In behalf of the Section I will ask for a rising vote of thanks to these gentlemen.

The meeting is now adjourned.

[Adjourned at 11.30 a. m.]

STUDIES IN ASTHMA*

XIX. The Nose and Throat In Five Hundred Cases of Asthma

BY FRANCIS L. WEILLE, M.D.†

IN the Anaphylaxis Clinic of the Massachusetts General Hospital a special study has been made of the nose and throat in asthma. Five hundred patients with asthma have been examined and treated and have been followed, in the great majority of cases, for a considerable length of time. Four hundred have been followed from six months to six years; three hundred and sixty-six for one or more years; and, two hundred and seven for three years or longer.

The patients examined and treated were not segregated from the remainder of the Anaphylaxis Clinic; the attention given the nose and throat was made a part of the patient's treatment, but not all of it. Major emphasis was placed upon the nose and throat therapy in some cases and in others such therapy constituted the sole treatment. The diagnosis and treatment of the cases was spread over a period of several years and an earnest effort was made to give all patients an adequate amount of time and effort in the solution of their problems.

Classification of the facts observed was accomplished by transferring the interesting points regarding each patient to a card system. These facts were then coded and tabulated by means of a business statistical machine owned and operated by the Harvard Medical School in its School of Public Health. This method not only discovers inaccuracies but lends itself toward ease in permitting corrections. The method may be recommended for its relative simplicity and for its flexibility in permitting further study of the same group at a later date. By means of the method employed it is possible to review five hundred and forty facts concerning each of the five hundred cases and to evaluate any desired observation in terms of any other observation included in the code.

It is said that statistics can prove anything. In the present study no effort was made to prove anything save the truth. The facts were observed and tabulated; then conclusions were drawn. The converse method was not employed.

The patients are classified according to age in chart 1. Almost one half were between thirty and fifty years. So few children of ten years or less were included that deductions regarding them are worthless. As regards the kind of asthma, two hundred and forty had extrinsic asthma, two hundred and fifty-five had intrinsic asthma and five had miscellaneous diagnoses—"unclassified", "chronic bronchitis and emphysema" and so forth. The methods employed in arriving at such diagnoses were similar to those employed in other reported Studies in Asthma from the Anaphylaxis Clinic[1] of the Massachusetts General Hospital.

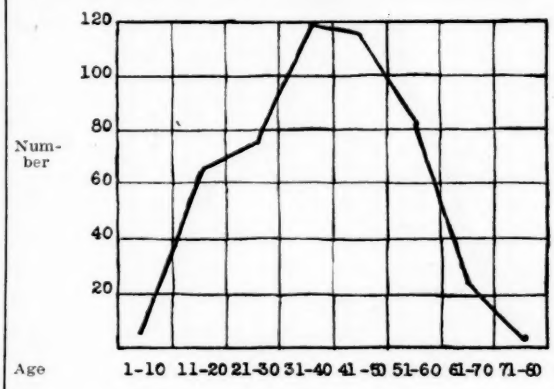

CHART 1.
Classification of Cases According to Age.

The more important nose and throat lesions in the group are presented in chart 2; these are simultaneously classified according to the kind of asthma. Pansinusitis is three times more common in intrinsic than in extrinsic asthma: less extensive sinusitis is, however, only a third more common. Nasal polypi were seen two thirds more often in intrinsic than in extrinsic asthma. The quick assumption that such polypi and the associated sinusitis alone

CHART 2

NOSE AND THROAT LESIONS

Lesions	Total	Extrinsic	Intrinsic	Miscellaneous
Pansinusitis (unilateral or bilateral)	46	11	34	1
Sinusitis—one or more (unilateral or bilateral)	316	136	180	0
Vasomotor rhinitis	223	111	109	3
Polypi	211	81	128	2
Surgically deviated septum	95	47	46	2
Severe chronic tonsillitis	35	15	20	0
Abscessed teeth	84	41	43	0

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

The expenses of this investigation were met from an anonymous donation known as the "M. G. H. Asthma Fund."

From the Anaphylaxis Clinic of the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary.

†Weille, Francis L.—Assistant Surgeon, Massachusetts Eye and Ear Infirmary. For record and address of author see "This Week's Issue," page 259.

"cause" asthma may be an easy fallacy in judging an incompletely studied patient. Septal deviations of severe degree are about equally distributed between the extrinsic and intrinsic cases. The problem of diseased teeth and tonsils is dealt with elsewhere.

Unstable nasal mucous membrane associated with vasomotor rhinitis or simulating this condition was observed in two hundred and twenty-three patients, almost equally in extrinsic and intrinsic asthma. One hundred and fifty-four cases complained of a thin watery nasal discharge while one hundred and thirty-two patients were distressed by severe sneezing. The commonness of mucous membrane instability in the nose suggests that similar pathology may be present in the tracheobronchial tree and be an important factor in precipitating the asthmatic attack. This point was made in a previous report on the basis of bronchoscopic observations.[2]

The frequency of sinusitis, as seen in three hundred and sixty-two of the five hundred cases, is striking. This observation should serve to make the rhinologist "allergy-minded" and the allergist "sinus-minded". It should be pointed out that many of the three hundred and sixty-two patients did not have severe sinusitis; merely a thickened membrane in the sinuses was extremely common, especially in the antrums and ethmoids. Two hundred and sixty of the patients had thickened membrane in at least one antrum, though some of the same patients had more severe sinusitis elsewhere. The hypothesis that thickened sinus mucous membrane is merely a part of the asthma and not the cause of it appears to be a reasonable one in view of mucous membrane instability observed throughout the respiratory tract in asthma and the probable labile character of such sinusitis, at least occasionally, as reported by Proetz.[3] The importance of such sinusitis lies in the fact that, when patients so affected have acute upper respiratory infections, the sinuses frequently become worse and their tendency is to keep the asthma going. Moreover, chronically thickened sinus membranes may become worse spontaneously and become polypoid and purulent. Regardless of cause and effect in relation to asthma, such degenerative changes place a further burden upon the patient's general health.

Severe sinusitis in the group studied was most frequently seen as a purulent antrum or as ethmoiditis with polypoid degeneration. Conversely, the polypoid antrum or purulent ethmoid has been less commonly observed. Combined purulent and polypoid sinusitis in the antrum or ethmoid was frequently seen.

CHART 3

RESULTS ON ASTHMA

Treatment	Total	Extrinsic Asthma				Intrinsic Asthma			
		Cured	Better	Same	Worse	Cured	Better	Same	Worse
No operation	290								
Intranasal antrum (one or both)	17	1	4	0	0	1	7	2	2
		Extrinsic: five				Intrinsic: twelve			
Radical antrum (one or both)	41	1	3	3	0	3	18	10	3
		Extrinsic: seven				Intrinsic: thirty-four			
Intranasal ethmoid (one or both)	30	1	1	1	0	2	17	6	2
		Extrinsic: three				Intrinsic: twenty-seven			
External ethmoid (one or both)	5	0	0	1	0	0	1	2	1
		Extrinsic: one				Intrinsic: four			
Sphenoids and external frontals (one or both)	7	0	0	1	0	1	4	1	0
		Extrinsic: one				Intrinsic: six			
Tonsillectomies	36	1	11	3	0	0	13	6	2
		Extrinsic: fifteen				Intrinsic: twenty-one			
Cases having extraction of abscessed teeth	59	1	14	5	3	2	22	7	5
		Extrinsic: twenty-three				Intrinsic: thirty-six			

The following table summarizes interesting sinus x-ray findings:

	Antrum (one or both)	Ethmoid (one or both)	Frontal (one or both)	Sphenoid (one or both)
Purulent sinusitis	62	30	11	9
Polypoid degeneration	31	45	17	17

The surgical treatment employed with the end-result evaluated in terms of asthma and its kind is summarized in chart 3. The principles utilized in the selection of cases for sinus surgery have been discussed in a previous report.[4] From the figures seen in the chart it will be observed that two thirds (66 per cent) of all sinus operations resulted in more or less improvement in the asthma and that such improvement was about the same whether the asthma was intrinsic or extrinsic. It will be observed that a higher percentage of improvement in asthma followed conservative nasal surgery

rather than very drastic efforts, such as external ethmoidectomy.

The local results are summarized in chart 4. In general it may be said that results in the nose were satisfactory and were accomplished by means of relatively conservative surgery.

THE TONSILS IN ASTHMA

Two hundred and fifty-nine of the five hundred patients had normal tonsils. One hundred and eighty-six had chronic tonsillitis, but only thirty-five of these patients had severe chronic tonsillitis. One hundred and nineteen members of the group had had a previous tonsillectomy; unfortunately numerous patients in this group had more or less troublesome tonsil remnants.

Tonsillectomy was advised and carried out in fifteen of the thirty-five severe cases and in twenty other patients. While this procedure was advised also in many additional instances, numerous patients were allowed to retain infected tonsils. Twenty-five members of the group undergoing operation showed improvement, but only one case was cured. Three additional patients were temporarily cured, but later became the same as before operation. In four cases the asthma was the same as before operation and in two patients it became worse.

These figures suggest that while some degree of improvement occurs in the asthma in a relatively high percentage of selected tonsillectomized cases, cure is rare.

THE TEETH IN ASTHMA

One hundred and thirty-three members of the total group of five hundred had x-ray films of the teeth. In every such case films of all the teeth were taken. In only eighty-four patients were abscessed teeth discovered and, of these, only fifty-nine had the abscessed teeth extracted. The following table summarizes the results in the asthma:

Asthma	Intrinsic	Extrinsic	Total
Cured	2	1	3
Better	22	14	36
Same	7	5	12
Worse	5	3	8

It will be observed that thirty-nine of the patients were improved or cured whereas twenty were the same or worse. This ratio is about two to one and is the same whether the asthma is intrinsic or extrinsic.

BRONCHOSCOPY IN ASTHMA

A group of thirty-six patients may be mentioned as having had bronchoscopy as a part of their treatment. The principles of selection of these cases have been described in a previous report[2] which included fifteen members of the group. Twelve of the patients were improved and three were cured. Six were temporarily cured, but later were the same as before treatment. The remaining cases were not benefited. During the period of observation nine patients died, five from asthma and the remainder from other causes. The high mortality in this group emphasizes the severity of the disease dealt with. A dramatic contrast was seen in two patients both of whom appeared moribund in an asthmatic attack. One was saved from drowning in her own viscous secretions by bronchoscopic aspiration of the tracheobronchial tree whereas the other died when only intense congestion of the mucous membrane, with little secretion, was found at operation.

The commonness of mucous membrane instability in the tracheobronchial tree in the group is striking and suggests itself as a factor in the production of asthmatic attacks. Such instability is more striking to the observer in the living patient than the hypertrophy of the smooth muscle of the bronchioles in the postmortem examination of the lung. The response of such mucous membrane to topical drug therapy varies, somewhat after the fashion of unstable nasal mucous membrane.

CHART 4

RESULTS IN NOSE

Treatment	Total	Extrinsic Asthma				Intrinsic Asthma				Unknown
		Cured	Better	Same	Worse	Cured	Better	Same	Worse	
No operation	290									
Intranasal antrum (one or both)	16	1	4	0	0	0	11	0	0	
			Total of five				Total of eleven			
Radical antrum (one or both)	35	0	3	0	0	2	24	3	3	
			Total of three				Total of thirty-two			
Intranasal ethmoid (one or both)	30	1	3	0	0	2	20	2	2	
			Total of four				Total of twenty-six			
External radical ethmoid (one or both)	5	0	0	0	2	0	3	0	0	
			Total of two				Total of three			
Frontals and sphenoids (one or both)	7	0	0	0	1	0	4	0	2	
			Total of one				Total of six			

An attempt to stabilize the bronchial mucous membrane by diathermy desiccation in one case was unsuccessful.

THE NONSURGICAL GROUP

It is interesting to observe the results in the asthma in a group of cases having no form of surgical treatment—whether drainage of sinuses, bronchoscopy, removal of polyps, teeth, tonsils and so forth. Two hundred and ninety of the five hundred patients constitute this group. One hundred and forty-two of these cases had slight to severe degrees of sinusitis; fifteen had pansinusitis. One hundred and forty-eight had clinically normal sinuses, confirmed by the x-ray examination in eighty-seven instances.

One hundred and sixty-four of the patients had normal tonsils, whereas forty-five had no tonsils and eighty-one had infected tonsils. Of the latter, twenty had severely infected tonsils. Forty-nine members of this group had normal teeth by x-ray examination, whereas twenty-five patients had abscessed teeth which were recognized by the same method, but which were untreated.

Chart 5 is a summary of the results in the asthma in the two hundred and ninety patients. About two thirds were improved or cured; the remainder were the same or worse. Since some members of the group had little or no pathology in the nose, throat or teeth, these figures do not make a fair comparison with the groups of patients treated surgically. It is to be emphasized that numerous cases included in the two hundred and ninety patients just described were directed to have more or less drastic treatment but failed to carry out such advice.

CHART 5

RESULTS IN ASTHMA

Treatment	Total	Extrinsic Asthma				Intrinsic Asthma				Unknown
		Cured	Better	Same	Worse	Cured	Better	Same	Worse	
No operation	290	14	94	36	9	8	52	37	5	
		Extrinsic: 153 cases				Intrinsic: 102 cases				35

MISCELLANEOUS OBSERVATIONS

Occasional patients included in the present study have complained that the asthma followed a nose or throat operation, such as tonsillectomy, submucous resection of the septum or some form of sinus drainage. It is interesting to contrast such cases with those who attribute improvement or cure of the asthma to similar surgical procedures. Not infrequently patients have been observed who ascribe improvement in well-defined extrinsic asthma to some form of nose and throat therapy, whereas medical opinion has explained the benefit as due to immunization against or removal of an allergen.

In certain cases a recurrent postnasal discharge has been associated with the onset of productive cough and wheezing; symptomatic relief from the postnasal discharge in these cases has accompanied relief from the asthma.

SUMMARY AND CONCLUSIONS

1. Facts not theories have been reported.
2. Patients should remain free of asthma for at least three years before being reported in the literature as cured.
3. Patients with intrinsic or extrinsic asthma may be improved or cured of asthma even though retaining a severe degree of sinusitis, infected tonsils or abscessed teeth. A focus may be present in extrinsic asthma without causative relationship to the asthma save by making the general condition of the patient worse.
4. Occasional cures of extrinsic asthma may result from the removal of an allergen whereas the patient ascribes the improvement to some form of nasal treatment.
5. If asthma is a disease of the parasympathetic nervous system, this may explain why some patients develop asthma after nose and throat operations and why others lose their asthma after similar operations.
6. Mucous membrane instability is seen throughout the respiratory tract in many asthma patients. Such instability in the tracheobronchial tree resembles strikingly that seen in the nose in two hundred and twenty-three of five hundred patients. Such instability in the tracheobronchial tree is a part of asthma; in the nose it is a part of the same underlying condition.
7. The fact that various methods of treatment produce nearly uniform results—two thirds better or cured, one third same or worse—suggests a spontaneous tendency toward remissions in asthma.
8. The frequent occurrence of sinusitis in allergic patients should make rhinologists "allergy-minded" and allergists "sinus-minded".
9. Conservatism in the selection of patients and in the choice of operative procedure is desirable in dealing with sinusitis in asthma.

REFERENCES

1. (a) Rackemann, Francis M.: Studies in asthma. I. A clinical survey of 1074 patients with asthma followed for two years. J. Lab. & Clin. Med. **12**:1185 (Sept.) 1927.
(b) Rackemann, Francis M.: Studies in asthma. II. An analysis of two hundred and thirteen cases in which the patients were relieved for more than two years. Arch. Int. Med. **41**:346 (March) 1928.
(c) Rackemann, Francis M., and Scully, Margaret A.: Studies in asthma. III. The use of vaccines in asthma and in "colds". New Eng. J. Med. **199**:314 (Aug. 16) 1928.
(d) Rackemann, Francis M., and Tobey, Harold G.: Studies in asthma. IV. The nose and throat in asthma. Arch. Otolaryng. **9**:612 (June) 1929.
(e) Rackemann, Francis M., and Colmes, A.: Studies in asthma. V. The clinical characteristics of hypersensitiveness. J. Allergy. **1**:2 (Nov.) 1929.
(f) Rackemann, Francis M.: Studies in asthma. VIII. The incidence and severity of asthma according to the season of the year. New. Eng. J. Med. **199**:1021 (Nov. 22) 1928.
(g) Rackemann, Francis M., and Colmes, A.: Studies in asthma. IX. Cough as a manifestation of human hypersensitiveness. J. A. M. A. **95**:192 (July 19) 1930.
(h) Rackemann, Francis M.: Asthma. XVI. Two hundred and thirteen "cured" patients followed up four years later. Arch. Int. Med. **50**:819 (Dec.) 1932.
2. Weille, Francis L.: Studies in asthma. XIV. Bronchoscopy in asthma. New Eng. J. Med. **205**:848 (Oct. 29) 1931.
3. Proetz, Arthur W.: Sudden allergic reactions localized in the antrum. Ann. Otol. Rhin. & Laryng. **39**:87 (March) 1930.
4. Weille, Francis L.: Studies in asthma. XVIII. The surgical treatment of chronic sinusitis in asthma. J. A. M. A. **100**:241 (Jan. 28) 1933.

FACTORS INFLUENCING THE DEVELOPMENT OF TUBERCULOUS INFECTION IN CHILDHOOD

BY ALLEN S. JOHNSON, M.D.*

THIS study was undertaken in an attempt to evaluate some of the factors supposed to influence the development of tuberculous infection in children. The group in question comprised 375 children under sixteen years of age who were examined at the Health Department Tuberculosis Dispensary in Springfield. Two hundred and eighteen children gave a definite history of contact with a known case of pulmonary tuberculosis; 157 noncontacts were presented for examination because of malnutrition, recurrent respiratory infections, or vague apprehension on the part of the parent. The examination of each child included a record of the age, sex, height, weight, temperature, cutaneous tuberculin reaction after forty-eight hours, x-ray of the chest, source of contact, period of exposure, and an appraisal of domestic hygiene as a result of personal investigation of the home.

The importance of exposure is well known. Opie[1] states that approximately 80 per cent of children of households in contact with persons having positive sputa had acquired infection within the first five years of life whereas of households in contact with a consumptive with a persistently negative sputum only 30 per cent are infected. Of approximately 48,000 grade school children examined in Massachusetts in 1930-31 about 25 per cent were found to be reactors.[2] As this includes some contacts, the incidence among noncontacts in the population at large is obviously lower. In table 1 it will be seen that only 21 per cent of the children who had never been exposed to a recognized case of tuberculosis showed a positive skin reaction whereas 57 per cent of all the contact children and 63 per cent of those exposed to positive sputa had positive skin reactions.

The sex of the child appeared to have little bearing on the susceptibility to tuberculous infection as has been pointed out by Amberson et al.[3] The slightly higher incidence of positive tuberculin reactions among the girls in this study is of little significance in view of the small series investigated.

It has been generally assumed that a tuberculous mother constitutes a greater risk to the child than an infected father because of her closer association with the offspring. Contrary to our expectation, it was found that 70 per cent of the children exposed to a tuberculous father had positive reactions whereas only 53 per cent of those with a tuberculous mother gave such a reaction (table 2). When a sibling or other relative living under the same roof was the source of contact, only 38 per cent of the exposed children had positive skin reactions. This was not due simply to the fact that more fathers than mothers might have had pos-

TABLE 1

RELATION OF EXPOSURE TO THE DEVELOPMENT OF TUBERCULOUS INFECTION IN CHILDREN

	Total Contacts	Contacts from Homes with Positive Sputa	Noncontacts
	218 (109 M) (109 F)	158	157 (68 M) (89 F)
Positive Tuberculin (cutaneous)	125 (60 M) (65 F)	100 (47 M) (53 F)	33 (17 M) (16 F)
Percentage	57.3	63	21
Positive Tuberculin Positive X-Ray (hilum shadows)	49 (24 M) (25 F)	36 (20 M) (16 F)	12 (6 M) (6 F)
Percentage	22.4	27.7	7.6

*Johnson, Allen S.—Visiting Physician, Tuberculosis Division, Springfield Health Department Hospital. For record and address of author see "This Week's Issue," page 259.

itive sputa as the same general relationship obtained when contact with cases of open tuberculosis were studied.

In table 3 an attempt was made to determine in which five-year period of its first fifteen years a child was the most vulnerable from the standpoint of tuberculous infection. The cases were divided according to the five-year period during which the maximum exposure occurred. There was necessarily some overlapping but our detailed knowledge of the past history of the infected parent, who had almost always been cared for in our clinic or hospital, enabled us to make certain generalizations. The contact children who had been exposed chiefly during the first five years of life did not show the highest incidence of positive skin reactions. The second and third five-year periods appeared to be distinctly more vulnerable and this same relationship held for exposure, both to all cases diagnosed as tuberculous by x-ray or sputum and to those actually having a positive sputum.

TABLE 2

RELATION OF SOURCE OF INFECTION TO TUBERCULOUS INFECTION

	To Father	To Father Having Positive Sputum	To Mother	To Mother Having Positive Sputum	To Siblings	To Siblings Having Positive Sputum
Number of Children Exposed	90	68	86	62	42	28
Positive Tuberculins	63	52	46	35	16	13
Percentage	70	76.5	53.5	56.5	38	46.6

TABLE 3

RELATION OF THE PERIOD OF MAXIMUM EXPOSURE TO THE DEVELOPMENT OF TUBERCULOUS INFECTION

Age Period of Maximum Exposure	Total Cases	Exposed to Positive Sputum	Positive Tuberculin	Exposed to Positive Sputum and Having a Positive Tuberculin	Percentage
0-6 years	112		60		53.5
		80		47	58.7
6-11 years	58		36		62.0
		44		30	68.0
11-16 years	48		29		61.5
		34		23	67.5

On the other hand, of all the contact children having positive skin reactions, the highest percentage was found to have received maximum exposure during the first five years of life (table 3a).) This does not invalidate the foregoing conclusion concerning the most dangerous time to be exposed, as the majority of contact children with negative reactions also experienced their maximum exposure during this same period. Adult pulmonary tuberculosis is a disease of early adult life during which period in the lives of their parents most children are born. Obviously children born into a home where tuberculosis exists will receive their maximum exposure during their first five years. Subsequently the parent is likely either to grow worse and leave home for a sanatorium or a cemetery, or to recover. In either case the contact is broken so that exposure of the child is unlikely to continue much more than five years.

TABLE 3a

Contact Children with Positive Tuberculin	Maximum Exposure	Per Cent	Maximum Exposure	Per Cent	Maximum Exposure	Per Cent
	0-6 years		6-11 years		11-16 years	
125	60	48	36	28.8	29	23.2
With Negative Tuberculin						
93	50	53.8	23	24.7	20	21.5

The older the child, the greater the chance of his having a positive reaction because his mere existence, irrespective of known exposure to tuberculous infection, increases his opportunities for contact with stray tubercle bacilli.[1] In 1910 Hamburger[4] showed that the percentage of positive reactors to the von Pirquet rose from 9 per cent at two years to 94 per cent at fourteen years. Opie[5] nearly twenty years later reported the results of intracutaneous tuberculin tests on 4000 school children. Thirty-seven per cent were infected at five years; 71 per cent at ten years; 80 per cent at fifteen years. Of 48,000 Massachusetts school children on whom the von Pirquet test was done in 1930-31, 20 per cent gave positive reactions at five years and 34.6 per cent at fifteen years.[6] A similar progressive increase in the incidence of positive tuberculin reactions in ascending age groups is seen in table 4 where they are also classified as contact and noncontact cases.

In table 5 is shown the relation between the

duration of exposure and the incidence of infection as indicated by a positive skin reaction. As might be expected, the longer the exposure, the greater the chance of infection. The fact that the children exposed four years or more did not show a higher incidence of infection than those exposed for two years may be due to the fact that many of the sources of contagion in the former group were old inactive cases that probably constituted a real danger during only a small part of the time in which the child was actually exposed. The sicker cases with the more active infection usually went away to a sanatorium or died within a year or two after the diagnosis was made, so that the contact was broken sooner than in the case of the chronic but relatively inactive patient.

TABLE 4

RELATION OF THE INCIDENCE OF TUBERCULOUS INFECTION TO THE AGE OF THE CHILDREN EXPOSED

Age Group	Total Cases	Positive Tuberculin Reactions	Per Cent
0-6 years	84	21	25
Contacts	50	19	38
Noncontacts	34	2	5.9
6-11 years	137	52	38
Contacts	76	44	58
Noncontacts	61	8	13
11-16 years	154	85	55
Contacts	92	63	68.5
Noncontacts	62	22	35.5

TABLE 5

RELATION OF THE DURATION OF EXPOSURE TO THE INCIDENCE OF POSITIVE TUBERCULIN REACTIONS

Years of Exposure	Number of Cases	Positive Tuberculin Reactions	Per Cent
1	118	55	46.5
2	33	25	75.7
3	18	12	66.5
4 or more	34	24	70.5

Tuberculosis has long been recognized as a disease of social and economic destitution. In table 6 is recorded our attempt to determine whether a child exposed to tuberculosis in an unhygienic environment ran a greater risk of infection than a child more fortunately situated. Class A homes implied neatness, cleanliness, a fair amount of sunlight and fresh air, and reasonable intelligence with evidence of hygienic consciousness on the part of the inmates. Class C homes were dirty, poorly situated, and inhabited by stupid or irresponsible people. Class B was intermediate between the two. The incidence of positive reactions among the noncontact children was similar in all three classes. This is to be expected, since poor domestic hygiene would not materially increase the danger of tuberculous infection in a home free of tuberculosis. On the other hand, we were surprised to find that the contact children from Class A homes had almost as high an incidence of positive reactions as those from Class C homes. One cannot conclude from this that good hygiene is unimportant. But it may be that other contributory factors, not fully recognized, exert a great deal of the influence usually attributed to domestic hygiene *per se*. This is in accord with the conclusions of Amberson et al.[3] that so far as general characteristics were concerned no one age, sex, social or economic group could be identified to include the greater portion of infection and that general health conditions were not illuminating so far as the chances of positive reactions were concerned.

TABLE 6

RELATION OF DOMESTIC HYGIENE TO THE INCIDENCE OF TUBERCULOUS INFECTION

Domestic Hygiene	Number of Cases		Positive Tuberculin Reactions	Per Cent
A	Contact	110	65	59
	Noncontact	75	15	20
	Total	185	80	43.2
B	Contact	55	26	47
	Noncontact	44	11	25
	Total	99	37	37.4
C	Contact	53	34	64
	Noncontact	38	7	18.8
	Total	91	41	45

The laity cherishes a belief that malnutrition predisposes a child to tuberculous infection. Amberson et al.,[3] however, have shown in a study of 1000 school children that only about 12 per cent of those infected were 10 per cent or more underweight. In table 7 it will be seen that 23

TABLE 7

RELATION BETWEEN STATE OF CHILD'S NUTRITION AND TUBERCULOUS INFECTION

	Number of Cases 10 Per Cent or More Underweight	Positive Tuberculins	Per Cent
Contact	38	24	63
Noncontact	55	13	23.3
Total	93	37	39.8
	Positive Tuberculins	**Number of Cases 10 Per Cent or More Underweight**	**Per Cent**
Contact	125	24	19.4
Noncontact	33	13	39.4
Total	158	37	23.4

per cent of the positive reactions were underweight and nearly 40 per cent of the underweight children had positive reactions. The higher figures here are probably due to the fact that our clinic was dealing with underprivileged children whom one might expect to be poorly nourished. As a matter of fact, approximately 25 per cent of all the children examined in the clinic and about 26 per cent of the negative reactions were 10 per cent or more underweight, using the Baldwin-Wood age, height, and weight tables as a standard.

This is admittedly a small group of children from which to draw conclusions. But this very feature has enabled us to study each child and his environment, as well as his source of contagion, in greater detail than is possible in large surveys dealing with thousands of cases. In order to check the possible errors due to the size of the group, a preliminary appraisal of the data was made halfway through the study. The same conclusions appeared warranted then as were reached after another 175 children had been examined a year later. Although this constancy of data may give one the temerity to draw conclusions it obviously lacks the support which a larger series would give.

SUMMARY

Close and persistent association with a case of pulmonary tuberculosis, especially one with a positive sputum, appears to be the most important predisposing factor in the development of tuberculous infection in children. It has not been possible to demonstrate that the age, sex, or nutrition of the child, the source of contagion, or the sanitary conditions in the home play an important part in conditioning tuberculous infection in the child.

Grateful acknowledgment is due to Miss Mary Carmody, R.N., and Mrs. Adelaide Perry, the clinic's social workers, whose careful tracing of contact cases has made this study possible.

REFERENCES

1. Opie, E. L.: Present concepts of tuberculous infection and disease. Am. Rev. Tuberc. **32:** 617 (Dec.) 1935.
2. Morgan: Mass. Dept. of Public Health, personal communication.
3. Barnard, M. W.; Amberson, J. B., Jr., and Loew, M. F.: Tuberculosis in adolescents; study of 1,000 school-children in New York City made under auspices of Bellevue-Yorkville Health Demonstration in 1930. Am. Rev. Tuberc. **23:** 593 (May) 1931.
4. Hamburger, F.: Allgemeine Pathologie und Diagnostik der Kindertuberkulose. Leipz. & Wien, 1910.
5. Opie, E. L.: The significance of advanced tuberculosis infection of school children. J. A. M. A. **95:** 1151 (Oct. 18) 1930.
6. Reinhardt: A comparative study of the relationship of childhood tuberculosis to adult tuberculosis. Mass. Dept. of Public Health, 1933.

MEDICAL PROGRESS

PROGRESS IN THE TREATMENT AND DIAGNOSIS OF SYPHILIS, 1935

BY AUSTIN W. CHEEVER, M.D.*

DURING the year 1935, nothing really new has been written regarding syphilis, though many excellent articles have been published. The general trend seems to be toward a study of the incidence, distribution, and prognosis of the disease with the hope of arriving at a better understanding of the problem. Of the drugs at our disposal mapharsen, which is the newest, is beginning to receive attention. This year also saw the best means of instructing the public, which heretofore had been closed to the mention of syphilis and gonorrhea, opened to instructive articles on these subjects. Several broadcasting stations, several good lay magazines, and some newspapers have laid aside their objections and it is hoped that more will follow suit.

Dublin[1] of the Metropolitan Life Insurance Company comments on the general trend of syphilis which he finds definitely downward in the white population in this country during the last two decades, while among the colored people the opposite is true. He urges greater concentration of attention to the syphilitic Negro population.

Chadwick,[2] Commissioner of Public Health of Massachusetts, reports from the prenatal clinics of Boston 1.19 per cent definitely positive and 1.99 per cent of positive and doubtful Hinton blood tests in the pregnant women. These are compared with Hinton's[3] 8.03 per cent positive and doubtful (less delicate Wassermanns) in 1923.

Nabarro[4] finds the incidence of congenital syphilis in England decreasing materially. The number of infants under one year of age has decreased one half, older children less. He emphasizes that, if syphilis were properly detected and treated in the expectant mother, congenital syphilis would be almost a thing of the past.

Cook[5] reports that in the United States Navy from 1914 to 1933 syphilis cases increased (in round numbers) from twenty to twenty-four per thousand, gonorrhea decreased from eighty-five to sixty-six per thousand, and chancroid decreased from forty-three to twelve per thousand. (A more delicate test for syphilis was

*Cheever, Austin W.—Assistant, Department of Dermatology and Syphilology, Harvard University Medical School. For record and address of author see "This Week's Issue," page 259.

used during the later part of this time, thus detecting more cases.) Usilton[6] by the one-day census method has made various estimates regarding syphilis and gonorrhea in the United States. The incidence is shown to be higher in the smaller cities than in the larger. About 493,000 patients are estimated to be constantly under treatment for syphilis in the United States. The incidence of fresh infections per year seems to be four per thousand for syphilis and eight per thousand for gonorrhea. The highest attack rate occurs between the ages of sixteen and thirty. The trend of syphilis seems to be upward with, however, a decrease in the prevalence rate for early syphilis, while the number of cases of gonorrhea actually under treatment has decreased, but it is believed that this is caused by neglect of treatment because of economic depression rather than an actual decrease in the number of infections. The Chief Medical Officer[7] of the Ministry of Health for England and Wales reports a continual decrease in syphilis while gonorrhea shows no evidence of decline.

Denmark shows a decrease of fresh infections from 4500 in 1919 to 700 in 1933. Lomholt[8] attributes these good results to an effective organization to combat syphilis as well as to effective treatment. The chief points are the following: (1) free treatment — about 70 per cent of these patients are treated by specialists who are paid by the city or the state; (2) compulsory treatment; and (3) punishment for knowingly infecting others with syphilis or gonorrhea. Jugoslavia[9] has recently enacted a law regarding the control of syphilis and gonorrhea. Among other provisions, it provides for the establishing of homes for unemployed women and girls. Institutions are also to be established for delinquent girls and for women who have been arrested for prostitution in an effort to restore them to a normal life.

Verano[10] et al. report of the activities of the Argentine League for Social Prophylaxis, the first of its kind in South America. They have been carrying on an active educational campaign. During the three years that this clinic has been functioning, about 70 per cent of the male candidates for marriage who have come for examination have had contagious stages of either syphilis or gonorrhea. The treatment of these patients has prevented a great amount of sickness and disability which otherwise would have occurred in their families. They are working toward a law requiring prenuptial examination.

An editorial[11] in the *Venereal Disease Information* comments sharply on the neglect of providing for the financing of venereal disease programs in a group of representative cities in the United States. It shows that expenditures for the control of communicable diseases amounted to 52.92 cents per capita in 1933, but for venereal control only 2.78 cents. The problem of syphilis and gonorrhea in the United States is a very complex one; and the solution of it will prove expensive. Unless health departments are more generous, but little can be expected in the campaign against syphilis and gonorrhea.

Gray and Cleveland[12] report two small epidemics of syphilis. One started in a seventeen-year old girl who gave it to three younger children, to two by sharing her gum with them and to the nursling by giving him food she had chewed. The mother subsequently developed a primary lesion of the nipple and in turn passed it to the father. The second group started with a father who had a primary lesion of the chin, gave it to his wife, and she apparently transmitted it to their three-year old child by feeding it food which she had masticated.

Morgan[13] reports sixteen cases of syphilis acquired through blood transfusions. There was no instance in which the donor had latent or chronic syphilis. He notes that many donors with chronic syphilis have failed to transmit the disease. Jones, Rathmell, and Wagner[14] review the literature on transfusional syphilis and report four additional cases. They urge greater care in excluding early syphilis in transfusion donors.

It has long been realized that the female with early syphilis may be rendered perfectly safe to a mate by two or three weeks of treatment which will heal the open lesions; thereafter she remains safe, if treatment is satisfactorily carried out. We have not known when the male becomes safe, nor do we yet, but a little light has been thrown on the problem by Greenbaum, Katz, and Rule[15] who injected the semen from twenty-five patients (five with acute and twenty with chronic syphilis) who had had some treatment for several weeks or months previous to the test, into the testicles of rabbits. After three months' observation, the inguinal lymph nodes were removed from the rabbits, ground up and injected into the testicles of other rabbits. At the end of three months these rabbits showed no macroscopic evidences of syphilis, and no spirochetes were found in material from the inoculated testicles by darkfield examination. This study demonstrates that we have had an exaggerated idea of the infectiousness of the semen of patients under treatment for syphilis and suggests a method of testing infectiousness that might be used before contemplated marriage in conjunction with the usual methods of examination.

Ballotta[16] feels that the only way to combat syphilis and gonorrhea effectively is to remove the stigma as far as possible and to induce the patient to report freely for treatment. He advocates having syphilis and gonorrhea consid-

ered as on the same footing with those diseases without moral implications. He urges that sickness insurance be applied to these diseases just as to any other contagious ones.

Sprague and White[17] find that there is a decrease in the incidence of aneurysm of the aorta from practically 0.2 per cent among the 52,000 patients admitted to the Massachusetts General Hospital between 1900 and 1909 to 0.08 per cent of the 75,000 patients in a similar ten-year period ending in 1934, and this, in spite of the improved x-ray facilities for diagnosis in the later period. Kampmeier[18] emphasizes the point, not always recognized, that race in itself is probably not a factor in the greater proportion of aneurysm in the black race, as compared with the white, but that the proportion of laborers is greater among the black.

Previously the work of Hinton and Berk[19] was reported showing that when the blood Hinton test is negative, the spinal fluid is almost certain to be negative. They found, however, 2 per cent doubtful positive spinal fluid tests in their series. Grund[20] reports four cases of positive spinal fluid with negative blood Hintons. Epstein[21] out of a large group of cases of central nervous system syphilis reports a few cases with positive spinal fluid findings and negative blood Hintons.

Menninger and Bromberg[22] from a study of all types of central nervous system syphilis find that about 30 per cent show a negative Wassermann reaction, whereas in 23 per cent neither the blood Kahn nor Wassermann was positive.

Beringer[23] reports in considerable detail an investigation carried out by a Russian-German expedition to Buriat-Mongolia to study the question of the course of syphilis in primitive people who have had little or no treatment. They wished particularly to investigate Wilmann's theory that neurosyphilis is a result of treatment. They feel that they have demonstrated that general paresis and tabes occur among primitive and untreated people and that modern treatment is not responsible for the development of neurosyphilis.

Hall[24] from a study of 191 cases of central nervous system syphilis finds that modern treatment shortens the period between infection and development of all types of late syphilis, when the late syphilis, because of inadequate treatment, does develop. Wile, Poth, and Barney[25] report a study with reference to precocious development of central nervous system syphilis. Patients admitted to the University of Michigan Medical School Clinic from July, 1925 to July, 1933 form the material upon which this study is based. The absence or inadequacy of treatment was a predominant factor in the precocious development of both dementia paralytica and tabes.

Goodman and Moore[26] carefully studied two groups of neurosyphilitic patients, those whose spinal fluid had become negative and those remaining positive. While they found some relationship between the reaction and the outcome, they found that the persistently positive reaction of the spinal fluid is not conclusive evidence of the subsequent progression of the condition, nor can the reversal of the reaction be used as the sole guide to the optimum duration of treatment.

Menninger[27–38] has covered every phase of juvenile paresis in a series of articles appearing in several journals, with extensive bibliography.

Solomon and Epstein[39] review the results of treatment with tryparsamide of eighty-one patients with dementia paralytica. Each had more than ten injections. Three of them were given more than 250. Of the eighty-one patients thirty-four were classed as arrested, that is, able to return to and continue their work. A completely normal spinal fluid was found in 37.5 per cent of the cases.

Tylecote[40] points out that in tabes dorsalis disability results chiefly from changes affecting the eyesight and the locomotion. The outlook depends very largely upon early diagnosis and adequate treatment. Much depends upon the intelligent interpretation of a suggestive sign or symptom—shooting pains, a perforating ulcer under one great toe, difficulty in beginning urination, cystitis, the sudden appearance of a squint, and sluggish or unequal pupils. A diagnosis of tabes must not be made, however, without verification. Ankle jerks are always lost before knee jerks, and if they are retained the apparent absence of knee jerks need not cause alarm. In the pre-ataxic stage, re-education may work wonders in preventing the onset of ataxia. The prevention of urinary infection is most important. When the patient complains of discomfort, regular tests of the urine and examination for retention must be made. A negative blood Wassermann is valueless in ruling out tabes dorsalis.

Belgrade and Wright[41] feel that in experienced hands the cisternal puncture, because of its lack of after-effects, is preferable to the lumbar puncture. Fluid is seldom contaminated with blood. Hospitalization is not necessary but it should not be undertaken without thorough preliminary study and practice on the cadaver. Cisternal puncture should not be attempted in elderly patients because of the added danger of injuring tortuous blood vessels, or in children, mainly because in them lumbar puncture is fairly easy and free from after-effects. Wilde[42] recommends cisternal puncture as a regular office procedure.

Under the title of "A New Syphilitic Dental Dystrophy" Karcher[43] describes a six-year

molar which is not of the mulberry type, on which there is no defect of enamel or early tendency to caries. The cusps, of normal size, are rolled in and without any shoulder at the crown and give the appearance of being clinched. He believes that the same condition has been very recently described in Germany by Kreyenberg and Schwisow.[44]

Kwazebart and Rosnowska[45] stress the great value of x-ray examination of the long bones in suspected congenital syphilitic infants, as they were able to make the diagnosis in some cases before there was any other evidence whatever. Parmelee and Halpern[46] show the superiority of x-ray examination over clinical and serologic in the detection in infants by a study of 104 born at Cook County Hospital. They found that 95 per cent could be diagnosed through the evidence of bone lesions. Periostitis almost never accompanies osteochondritis in the newborn. It becomes moderately frequent by the sixth week and is present in more than half of the cases showing osteochondritis by the third month. Periostitis very rarely occurs as the only change.

Ingraham[47] found that darkfield examination of the umbilical vein was positive in nineteen of eighty-five living offspring of syphilitic mothers. This is very valuable just at the time when other clinical and laboratory aids are likely to fail. He points out that a negative darkfield does not rule out syphilis. Because the method is rather cumbersome, it is practical only when the mother is known to have syphilis.

Roberts[48] in collaboration with McCord has studied one thousand infants born of syphilitic mothers. Of 563 treated mothers 17.7 per cent produced syphilitic offspring. This is compared with 65 per cent syphilitic offspring from 317 untreated mothers. If the amount of treatment was carried up to nine or more injections of neoarsphenamine, only 5.3 per cent produced syphilitic children.

Coppolino[49] reports 100 per cent morbidity of the offspring of untreated syphilitic primiparas as compared with 20 per cent for treated mothers. He is convinced that infants born of either treated or untreated mothers need no treatment provided there are no clinical or x-ray evidences at one month of age and the Wassermann is negative.

McCord[50] is quite sure that the reliability of the blood test is not affected by pregnancy. The blood test must be a routine part of antepartum care regardless of the activity of the disease. Antepartum treatment assures a healthy baby in 95 per cent of the cases. The best results occur with more than ten injections. There seems to be no condition in medicine that returns such huge dividends in life and health with such a small output of energy and money as that seen in the prevention of congenital syphilis.

In a study of ten thousand autopsies performed in Milan, Frates[51] found 403 cases in which syphilis had been diagnosed during life; of these only 178 showed evidence of syphilis at autopsy. About 30 per cent had not been diagnosed during life; by far the largest percentage was syphilis of the heart and blood vessels, making a total of 67.7 per cent. There were 13.8 per cent of syphilis of the central nervous system and 12.6 per cent of congenital syphilis. Other evidences were very few in number.

Neymann[52] has translated an article by Bessemans et al. of Belgium in which they describe a technique which they have worked out for counting spirochetes in lymph glands and in syphilomas.

Jahnel[53] has studied the effect of hibernation on experimental syphilis given just before the hibernating season. In the spring, syphilis was not present. This observation cannot be applied directly to man but he wonders if there may be some internal secretion factor in the problem which can be utilized.

Spangler[54] finds that a moderate degree of eosinophilia, especially when associated with a lymphocytosis, occurring even periodically in an apparently nonallergic individual whose intestinal tract is free from ova and parasites, warrants the taking of blood tests and a persistent search for clinical evidence of syphilis. The literature is reviewed with a list of thirty references.

Kitamura[55] has studied the spirochetes of syphilis and frambesia through a "comparative ocular" by means of which half of the field shows organisms of one sort and half shows the other. He finds them absolutely indistinguishable morphologically.

As contradictory reports have been published on the value of serological tests on the blood of cadavers, Bertolozzi[56] tested fifty cadavers. He found that all cases of syphilis were revealed but that false positives occurred as well, making the test of little medicolegal value.

Nelson[57] in a reply to a correspondent who thought that some confusion may have been created by the substitution of the Hinton test for the Wassermann in the laboratory of the State Department of Health, says that recent evaluation of serum tests by the United States Public Health Service[58] places the Hinton test among the most nearly specific of any in use in this country. No laboratory test is infallible but it is apparent that the physician is far less likely to be in error if he depends upon the results of repeated Hinton tests than the less specific Wassermann.

"The Department suggests that physicians accept the Hinton test as one of the most specific tests available, in that it is falsely positive with extreme rarity and detects syphilis

as well as, if not more frequently than, any other test in use. It should not be suspected of nonspecificity by comparison with the Wassermann or any other test. Its specificity has been studied against known infections and known freedom from infection for many years."

Chargin, Leifer, and Hyman[59] have treated by continuous intravenous drip method twenty-five patients with early syphilis. They gave an average of 4 grams of neoarsphenamine per patient over a period of five days. No serious reactions were noticed. They warn that this is only a preliminary study and the method should not be adopted except under ideal conditions.

Heuck[60] and Vonkennel[61] report experimental and clinical use of gold in conjunction with arsphenamine and bismuth in the treatment of syphilis. They feel that this combination has promise.

Jordan and Osborne[62] find that patch tests of patients for sensitivity to the arsphenamines are unreliable. Robinson[63] found, from careful patch testing, that the detection of arsphenamine sensitivity by that method has no value, since it is sometimes positive in normal individuals and often negative in those known to be arsphenamine-sensitive.

Induced by a favorable report on oral bismuth which had followed careful study[64] of numerous bismuth compounds, Kolmer,[65] having determined that the toxicity of water-soluble potassium bismuth tartrate by mouth is very low for rabbits and rats, found it therapeutically valuable in the human being. He recommends it in the treatment of occasional cases of syphilis especially in those unable to pay for intramuscular injections or those who are traveling or who for social reasons must avoid the detection of injection marks. The dosage is three to six grains a day. He believes that oral bismuth is superior to mercury in any of its various forms being less toxic and more spirocheticidal.

Mulzer and Serefis[66] used a complex liver-bismuth salt called bismutrat in forty-nine cases including all stages of syphilis. They felt that their results were equal to and in some cases better than results in cases that received intramuscular bismuth. Wise and Sulzberger,[67] Editors of the *Year Book of Dermatology and Syphilology*, consider this method of treatment promising and state that one of them, with others, is carrying on studies in the rabbit and the human being.

McCarthy and Dexter[68] carefully studied for bismuth deposits the mouths of about five hundred dentulous patients receiving intramuscular injections of bismuth salicylate. The incidence was about 80 per cent. Tartar accumulation was an important factor. In edentulous mouths the incidence was 9 per cent. There were only two cases of stomatitis in the series. In private patients, whose mouths are better cared for, the incidence was very low, about 5 per cent.

Kolmer[69] found daily inunction of rabbits with potassium bismuth tartrate to be without demonstrable curative value.

Oliver and Crawford[70] conclude from parallel series of cases treated with bismuth salicylate and iodo-bismuthate of quinine that the results suggest the superiority of the latter as regards both results and freedom from complications.

Sollmann, Cole et al.[71] report on the excretion of oral mercury which they believe began to be used therapeutically before 1535. They found that the excretion and presumably therefore the absorption and effective concentration in the tissues from the oral administration of 0.2 gram of mercury with chalk daily and .016 gram of protoiodide of mercury four times a day were nearly the same as obtained from 50 per cent mercurial ointment. Whereas from .015 gram of corrosive sublimate a day, the excretion was between one fourth and one third that with the administration of .065 gram of protoiodide a day. This difference corresponds closely to that in the content of mercury, but the excretion with this dosage of corrosive sublimate is probably below the desirable therapeutic level.

Sollmann, Cole et al.[72] give their conclusions regarding a series of studies on mercurial inunctions as determined largely by urinary excretion. Inunctions with metallic mercury secure a smoothly ascending concentration of the drug which declines rather slowly on discontinuance of medication. The amount excreted fairly parallels the concentration of the ointment. Cleansing the skin after inunction decreased the amount excreted by nearly one half. The oleate is colorless, therefore cleaner, easier to rub in, as well or better absorbed, and definitely advantageous, if inunctions are to be used. Calomel ointment is ineffective.

Arsenoxide was studied by Paul Ehrlich before his discovery of "606" and was considered very potent but dangerously toxic. It has since been better purified and used in both experimental animals and the human being under the name of mapharsen. Gruhzit[73] from extensive tests of this drug in laboratory animals found that by mouth it had no protective value against syphilis, that its therapeutic value was equal intramuscularly and intravenously in single doses. The therapeutic index of mapharsen and neoarsphenamine was about equal but with repeated doses the former appeared superior to the latter. One milligram of mapharsen was shown to have higher therapeutic value than ten milligrams of neoarsphenamine.

Foerster, McIntosh et al.[74] report on their use in the human being of arsenoxide, now man-

ufactured under the name of mapharsen. They gave the drug intravenously to eighty patients with syphilis exclusive of neurosyphilis to a total of 2117 injections. Spirochetes disappeared usually within twenty-four hours from open lesions. Healing was as rapid as with arsphenamine. Wassermann reactions became negative in nearly all cases of early syphilis, but returned positive in half the cases. Clinical relapse and serologic relapse were observed most often after irregular or short periods of therapy during the first half year with long lapses in treatment. Nitritoid reactions did not occur. The drug was usually well tolerated. Jaundice developed in four cases and accentuation of renal impairment was observed in four others. The authors feel that an extensive study of this drug for a period of years is justified. In the discussion Cole, from a study of one thousand injections, conservatively recommended the drug especially for its absence of nitritoid reactions and stated that he was pleased with its action. Wile agreed with Cole in his comments. Stokes made the point that reactions are a powerful factor in causing patients to discontinue treatment and that even if mapharsen should not prove so efficient, it is worth further study for its lack of reactions, if for no other reason.

Under the name of arsenoxide, Raiziss and Severac[75] of the Dermatological Research Laboratories at Philadelphia, compare this drug with neoarsphenamine. In rats and rabbits they found neoarsphenamine about five and one half times as effective in both its trypanocidal and spirocheticidal effect. In experimental rabbit syphilis the maximum tolerated dose and the minimum curative dose for arsenoxide are practically identical, which suggests that the clinical use of this drug in adequate curative doses might be fraught with considerable danger.

"Fixed" eruptions due to the trivalent arsenicals, while very uncommon, have been occasionally reported. Kemp and Menninger[76] report a case of "fixed" eruption due to tryparsamide of which they believe only one other case has been reported. It was not a contraindication to further treatment.

Tryparsamide has been supposed to be singularly free from reactions. The only one which has received much attention is disturbance of the optic fields. Hoverson[77] calls attention to mental derangements which occur during the use of tryparsamide and usually quiet down when the drug is discontinued. The psychosis is abrupt in onset and is usually a delirium often with vivid hallucinations of either the auditory or visual type, occasionally both, and fear reactions with restlessness and overactivity.

Muncy[78] from a study of eyegrounds in neurosyphilis concludes that: (1) the presence of contracted chromatic fields previous to the administration of tryparsamide is not a contraindication to its use; (2) contraction of the color fields in patients with positive reaction of the blood is a presumptive sign of beginning neurosyphilis.

Simpson[79] feels that after using the Kettering hypertherm for inducing fever there is evidence that artificial fever therapy fortifies and intensifies the activity of antisyphilitic chemotherapeutic agents.

Moore and Solomon[80] give a good review of Ulrich von Hutten's writings early in the sixteenth century. He is credited with first having discovered "syphilitic paralysis". They quote numerous interesting passages.

Saito[81] states that the most common place for extragenital chancres in Japan is on the breasts making up 36.4 per cent in his series. Fingers come next with 12.7 per cent, lips 10.9 per cent, and eyelids 5.5 per cent.

Fales and Reinhard[82] report that syphilis is the most costly of communicable diseases. They state that although measles was epidemic in the city of Baltimore last year, a three-year comparison of the incidence of reported measles and reported syphilis shows that for 18,905 cases of measles, a high mark, there were 13,032 cases of syphilis. No other communicable disease could have such a high incidence without arousing the medical profession, the health departments, the city authorities, and the entire citizenry to action. Unlike measles, which is normally a brief and passing disease, syphilis accounts for 10 per cent of the deaths from heart disease, the leading cause of death, and for 12 per cent of persons in institutions for the insane. The city of St. Louis spends between two and two and one-half million dollars annually on the care and treatment of those having syphilis and gonorrhea, says the report, and Baltimore undoubtedly spends a similar amount. A thoroughgoing public health campaign against these costly and destructive diseases is urged.

An editorial[83] in the *Venereal Disease Information* calls attention to the small amount of notice that seems so far to have been paid to good epidemiologic efforts toward eradicating syphilis. The work of Munson[84] in New York and Smith and Brumfield[85] in Virginia and the New York program which is being planned by Parran[86] show that beginnings have been made in this type of work in this country, but it has not been utilized to anything like the extent that it should be. It is true that it requires money, an organized effort, and an especially trained personnel for the work; but it would be cheap at any price, as the cost would be much less than that of caring for an increasing number of patients with syphilis. The policy of preventing this dis-

ease instead of paying for its consequences would greatly reduce the tax burdens of the future. In an epidemic of smallpox or typhoid fever, effort is immediately directed to tracing the source. Such investigative work is equally important in syphilis and gonorrhea. Every health officer should realize the fact that these diseases are communicable and that there can be no control of a communicable disease without investigation of sources of infection.

REFERENCES

1. Dublin, Louis I.: Ven. Dis. Inform. **16**:72 (March) 1935.
2. Chadwick, Henry D.: J. Social Hyg. **21**:195 (April) 1935.
3. Hinton, W. A.: Am. J. Syph. **7**:155 (Jan.) 1923.
4. Nabarro, David: J. Roy. San. Inst. **56**:172 (Oct.) 1935.
5. Cook, S. S.: U. S. Nav. M. Bull. **33**:405 (July) 1935.
6. Usilton, Lida J.: Ven. Dis. Inform. **16**:147 (May) 1935.
7. Social hygiene in England and Wales: Health News. **11**:702 (Dec. 17) 1934. Quoted in Ven. Dis. Inform. **16**:166 (May) 1935.
8. Lomholt, Svend: Dermat. Wchnschr. **100**:173 (Feb.) 1935.
9. The Jugoslavian law for the control of venereal disease. Mitt. d. deutsch. Gesellsch. z. Bekämpf d. Geschlechtskr. **32**:181 (Nov.-Dec.) 1934. Quoted in Ven. Dis. Inform. **16**:107 (April) 1935.
10. Verano, Alfredo F.: Semana méd. **41**:1791 (Dec. 6) 1934 and **42**:310 (Jan. 24) 1935.
11. Editorial: Ven. Dis. Inform. **16**:223 (July) 1935.
12. Gray, A. L., and Cleveland, W. H.: Am J. Pub. Health. **25**:49 (Jan.) 1935.
13. Morgan, Hugh J.: Am. J. M. Sc. **189**:808 (June) 1935.
14. Jones, H. W., Rathmell, T. K., and Wagner, Charles: Am. J. Syph. & Neurol. **19**:30 (Jan.) 1935.
15. Greenbaum, S. S., Katz, S., and Rule, A. M.: Am. J. Syph. & Neurol. **19**:210 (April) 1935.
16. Ballotta, F.: Arch. di antropol. crim. (supp.) **55**:58 (Jan.) 1935.
17. Sprague, Howard B., and White, Paul D.: J. A. M. A. **105**:1391 (Nov. 2) 1935.
18. Kampmeier, R. H.: Colorado Med. **32**:104 (Feb.) 1935.
19. Hinton, W. A., and Berk, Arthur: Am. J. Syph. & Neurol. **18**:92 (Jan.) 1935.
20. Grund, J. L.: Arch Dermat. & Syph. **32**:569 (Oct.) 1935.
21. Epstein, Samuel H.: New Eng. J. Med. **212**:495 (March) 1935.
22. Menninger, W. C., and Bromberg, Leon: J. Lab. & Clin. Med. **20**:698 (April) 1935.
23. Beringer, K.: Arch. f. Psychiat. **103**:359 (June) 1935.
24. Hall, Thomas B.: South. M. J. **28**:631 (July) 1935.
25. Wile, Udo J., Poth, Duncan O., and Barney, Burton F.: J. A. M. A. **105**:1329 (Oct. 26) 1935.
26. Goodman, M. J., and Moore, J. E.: Arch. Int. Med. **55**:826 (May) 1935.
27. Menninger, W. C.: Am. J. Syph. & Neurol. **18**:486 (Oct.) 1934.
28. Idem: Arch. Int. Med. **55**:626 (April) 1935.
29. Idem: J. Nerv. & Ment. Dis. **81**:489 (May) 1935.
30. Idem: Arch. Neurol. & Psychiat. **34**:243 (Aug.) 1935.
31. Idem: Ann. Int. Med. **8**:1287 (April) 1935.
32. Idem: Am. J. Syph. & Neurol. **19**:88 (Jan.) 1935.
33. Idem: Am. J. Psychiat. **91**:1413 (May) 1935.
34. Idem: Psychoanalyt. Rev. **23**:76 (Jan.) 1936.
35. Idem: J. Lab. & Clin. Med. **20**:806 (May) 1935.
36. Idem: Am. J. Syph. & Neurol. **19**:238 (April) 1935.
37. Idem: Am. J. Syph. & Neurol. **19**:257 (April) 1935.
38. Idem: Arch. Path. **19**:316 (March) 1935.
39. Solomon, Harry C., and Epstein, Samuel: Arch. Neurol. & Psychiat. **33**:1216 (June) 1935.
40. Tylecote, Frank E.: Lancet **1**:1117 (May) 1935.
41. Belgrade, Joseph T., and Wright, Carroll S.: Am. J. Syph. & Neurol. **19**:344 (July) 1935.
42. Wilde, H.: Med. Klin. **31**:850 (June) 1935.
43. Karcher, Edward W.: Arch. Dermat. & Syph. **31**:861 (June) 1935.
44. Kreyenberg, G., and Schwisow, W.: Ztschr. f. d. ges. Neurol. u. Psychiat. **127**:188, 1930.
45. Kwazebart, L., and Rosnowska, B.: Rev. franç. de dermat. et de vénéréol. **11**:195 (April) 1935.
46. Parmelee, A. H., and Halpern, L. J.: J. A. M. A. **105**:563 (Aug. 24) 1935.
47. Ingraham, Norman R., Jr.: J. A. M. A. **105**:560 (Aug. 24) 1935.
48. Roberts, M. Hines: J. Pediat. **6**:271 (Feb.) 1935.
49. Coppolino, John F.: Am. J. Obst. & Gynec. **29**:714 (May) 1935.
50. McCord, James R.: J. A. M. A. **105**:89 (July 13) 1935.
51. Frates, Achille: Clin. med. ital. **65**:1015 (Nov.) 1934.
52. Neymann, C. A.: Am. J. Syph. & Neurol. **19**:161 (April) 1935.
53. Jahnel, Franz: Arch. f. Dermat. u. Syph. **171**:187 (Jan.) 1935.
54. Spangler, Ralph H.: J. Lab. & Clin. Med. **20**:733 (April) 1935.
55. Kitamura, S.: Dermat. Ztschr. **71**:61 (April) 1935.
56. Bertolozzi, Menenio: Gior. di batteriol. e immunol., Torino. **13**:669 (Oct.) 1934.
57. Nelson, N. A.: New Eng. J. Med. **213**:381 (Aug. 22) 1935.
58. Cumming, H. S., et. al.: J. A. M. A. **104**:2083 (June 8) 1935.
59. Chargin, Louis, Leifer, W., and Hyman, H. T.: J. A. M. A. **104**:878 (March 16) 1935.
60. Heuck, W.: Arch. f. Dermat. u. Syph. **172**:75 (Aug.) 1935.
61. Vonkennel, J.: Arch. f. Dermat u. Syph. **172**:80 (Aug.) 1935.
62. Jordan, James W., and Osborne, E. D.: New York State J. Med. **35**:210 (March) 1935.
63. Robinson, Harry M.: South. M. J. **27**:845 (Oct.) 1934.
64. Serefis, S.: Arch. f. Dermat. u. Syph. **171**:1 (Dec.) 1934.
65. Kolmer, J. A.: Arch. Dermat. & Syph. **31**:9 (Jan.) 1935.
66. Mulzer, P., and Serefis, S.: München. med. Wchnschr. **81**:1525 (Oct. 5) 1934.
67. Wise, Fred, and Sulzberger, Marion B.: Year Book Dermat. & Syph., 1935, p. 667.
68. McCarthy, F. P., and Dexter, S. O., Jr.: New Eng. J. Med. **213**:345 (Aug. 22) 1935.
69. Kolmer, J. A.: J. Chemotherapy. **12**:278 (Oct.) 1935.
70. Oliver, E. Lawrence, and Crawford, G. Marshall: New Eng. J. Med. **212**:297 (Feb. 14) 1935.
71. Sollmann, T., Cole, H. N., et al.: Arch. Dermat. & Syph. **31**:15 (Jan.) 1935.
72. Idem: Arch. Dermat. & Syph. **32**:242 (Aug.) 1936.
73. Gruhzit, O. M.: Arch. Dermat. & Syph. **32**:848 (Dec.) 1935.
74. Foerster, O. H., McIntosh, R. L., et al.: Arch. Dermat. & Syph. **32**:868 (Dec.) 1935.
75. Raiziss, George W., and Severac, Marie: Am. J. Syph. & Neurol. **19**:473 (Oct.) 1935.
76. Kemp, J. E., and Menninger, W. C.: Am. J. Syph. & Neurol. **19**:195 (April) 1935.
77. Haverson, Emil T.: Am. J. Syph. & Neurol. **19**:217 (April) 1935.
78. Muncy, William M.: Rhode Island M. J. **18**:9 (Jan.) 1935.
79. Simpson, Walter M.: J. A. M. A. **105**:2132 (Dec. 28) 1935.
80. Moore, Merrill, and Solomon, Harry C.: Arch. Dermat. & Syph. **31**:692 (May) 1935.
81. Saito, K.: Jap. J. Dermat. & Urol. **37**:291 (March) 1935.
82. Fales, W. Thurber, and Reinhard, Ferdinand O.: Sight-Saving Rev. **5**:156 (June) 1925.
83. Editorial: Ven. Dis. Inform. **16**:347 (Oct.) 1935.
84. Munson, William L.: Am. J. Pub. Health. **23**:797 (Aug.) 1933.
85. Smith, Dudley C., and Brumfield, William A., Jr.: J. A. M. A. **101**:1955 (Dec. 16) 1933.
86. Parran, Thomas, Jr.: Ven. Dis. Inform. **16**:303 (Sept.) 1935.

DO YOU KNOW?

When Thomas Jefferson was president he actively supported the movement to establish smallpox vaccination.

In 1935 there were more than 8,000 cases of smallpox in the United States. This was half again as many cases as were reported in 1934. Yet vaccination has been practiced in this country since 1800 and is a certain way to protect the individual and to prevent the disease spreading to others. The northwestern states account for 5,000 of these cases: Washington, Idaho, Montana, Wyoming, Colorado, South Dakota, Nebraska and Kansas.

One out of every five white babies born will eventually die of some form of heart disease.—*Public Relations Bureau, Medical Society of the State of New York, 2 East 103d Street, New York, N. Y.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22321

PRESENTATION OF CASE

First Admission. A twelve year old schoolgirl was admitted complaining of stiff joints and diarrhea.

About one year before admission the patient developed a loose watery diarrhea with eight to nine movements daily unaccompanied by fever or vomiting. Two weeks after the onset blood was noted in the stool for three days. There was occasional tenesmus, vague abdominal pain, and the patient's weight dropped gradually from 78 pounds to 38 over a period of several months but rose again to 58 pounds at admission. After two and a half months she entered another hospital where a proctoscopic examination showed congestion of the mucous membranes and many scattered points of oozing. She received palliative and supportive treatment without subsidence of her diarrhea. While at the hospital she developed furunculosis and numerous muscular contractures. She was discharged after eight and a half months slightly improved and came to this hospital for further care.

Physical examination showed a poorly nourished child with marked pallor of the skin and mucous membranes. Numerous scars of previous abscesses were noted on the thighs, arms, and face, and there were draining abscesses on the left ankle and thigh. Both knees were fixed in flexion. There was some limitation of motion of the hips but the other joints were negative. The remainder of the examination was negative.

The temperature was 99.4°, the pulse 110. The respirations were 24.

Repeated urine examinations showed occasional sediment loaded with pus cells but were otherwise negative. The stools contained an abundance of mucus and occasionally large amounts of pus and blood. No parasites or ova were found. The blood showed a red cell count of 4,800,000, with a hemoglobin of 25 per cent. The white cell count was 12,800. Von Pirquet and intradermal tuberculin tests were negative.

A barium enema showed extremely rapid filling of the colon. There was no visible peristalsis or haustration. The colon was small, smooth and of even density throughout. X-ray examination of the joints showed some atrophy of the bones but no significant joint changes.

During her hospital stay the patient's temperature ranged between 98° and 100° irregularly and the diarrhea continued unchanged. Orthopedic treatment produced some improvement of the contractures. She received several transfusions and palliative care without subsidence of the diarrhea and on the fifty-first day an ileostomy was performed. At operation the entire colon was pallid, thickened, and contracted without normal haustrations. The cecum was not contracted but its walls were thickened and injected. The ileum appeared to be normal. Colonic irrigations through the ileostomy wound were performed thereafter. The patient showed slight improvement although the melena continued. After six months she was discharged to a convalescent home.

Second Admission, six months later, for further orthopedic care.

During the interval she had been fairly well. The ileostomy stoma functioned readily and only occasionally were pus and blood discharged from the rectum. She was unable to walk without assistance.

Physical examination was essentially unchanged. Limitation of extension of both thighs and knees persisted.

Traction was attempted but was discontinued promptly with the belief that the patient's general condition did not warrant further treatment. She was discharged on the fifth day.

Third Admission, one month later.

A week before re-entry the patient developed swelling, dark discoloration, and pain in the left knee and ankle. The right knee and ankle were similarly affected but to a less degree. The swelling slowly subsided but the joints remained painful.

Physical examination showed an emaciated girl with dry scaly yellowish skin and considerable muscle atrophy. The tongue showed roughened edges, hypertrophied papillae, and small whitish patches. The heart was slightly enlarged to the left and showed an occasional extrasystole. A widely transmitted systolic murmur was heard over the apical region. The lungs were negative. The right wrist, both ankles, and the left knee were swollen, moderately tender, and showed limitation of motion.

The temperature varied between 100° and 103° and the pulse remained above 120.

Examination of the urine was negative. The blood showed a red cell count of 4,100,000, with a hemoglobin of 60 per cent. The white cell count was 18,720, 74 per cent polymorphonu-

clears. A Wassermann test of the blood was negative. Colonic irrigation returned washings which contained both red and white blood cells microscopically. After seven days the patient was discharged slightly improved.

Fourth Admission, five years later.

Following discharge the patient had periods of partial remission but most of her previous symptoms returned intermittently. Three weeks before returning to the hospital she again developed multiple joint pains and three days prior to entry a painful swelling appeared at the outer aspect of the right knee. This rapidly increased in size, ruptured, and discharged thick purulent material. Other abscesses appeared on both legs subsequently.

Physical examination showed multiple ragged scars in different portions of the body and extremities. There was marked muscular atrophy of the extremities and many joints were ankylosed and tender. The ileostomy stoma was surrounded by an area of reddened induration and discharged considerable pus. There were several cutaneous abscesses on both legs.

The temperature was 100°, the pulse 120. The respirations were 20.

Examination of the blood showed a white cell count of 44,000. The abscesses were drained. Blood cultures and material from the abscesses produced no growth on culture. Culture of the rectal mucosa showed hemolytic streptococci.

X-ray examination of the bones of the feet showed marked bone atrophy with obliteration of the joint spaces.

The patient was treated surgically and by dietary measures and received colonic irrigations with various types of streptococcus antitoxic sera. She was discharged on the thirty-sixth day.

Fifth Admission, six and a half years later, at the age of twenty-four.

The patient remained comparatively well following her last discharge and no complaints were noted. Two days before returning to the hospital she suddenly developed generalized steady abdominal pain, there having been a bloody discharge from the ileostomy for several days. She took some sodium bicarbonate which was promptly vomited but the pain was slightly lessened. There was marked anorexia. On the day of entry the pain again became very severe and she vomited three or four times. She believed that the usual amount of material was passed through her ileostomy during this acute illness.

Physical examination showed a fairly well nourished young woman moaning with pain. The heart and lungs were negative. The abdomen was slightly distended and the ileostomy appeared to be functioning normally. There was moderate generalized abdominal spasm, more marked on the right side, flank, and right costovertebral angle. In these regions there was marked tenderness and rebound tenderness. Rectal examination could not be done because of tightness of the sphincter.

The temperature was 103°, the pulse 120. The respirations were 25.

Examination of the urine was negative. The blood showed a red cell count of 4,000,000, 88 per cent hemoglobin. The white cell count was 33,700.

Shortly after entry a laparotomy was performed. Through a left paramedian incision the peritoneum seemed free of adhesions but the pelvis was filled with odorless turbid brownish fluid. The small intestine was negative but the segment between the ileostomy and cecum was not identified. The tip of the cecum was tremendously thickened, injected, tense and firm. What was believed to be an area of ascending colon was shiny and purplish in color and there was some adherent fibrin. The first portion of the transverse colon was thickened, pale and contracted. A right flank incision was then made and the cecum and ascending colon thus exposed were tense, firm and light pink in color with no evidence of necrosis. The entire segment of bowel was fixed. An area of cecum and ascending colon was packed off in order to produce extraperitonealization. On the following day a cecostomy was done and a few ounces of pus evacuated from the bowel. The temperature gradually subsided, the white blood cell count dropped to 6,000, and the abdomen became soft. The pericolic packs were gradually removed and the patient was discharged on the eighteenth hospital day.

Final Admission, two months later, thirteen years after the initial entry.

The patient, now twenty-four years of age, attended the Out-Patient Department several times. She felt well until two weeks before reentry, when she developed a dull heavy feeling in the epigastrium which occasionally radiated beneath the sternum to the precordium. Sharp pains of a few minutes' duration were occasionally superimposed upon the dull discomfort but there was no vomiting, chills, fever, or abnormal bowel movements. Two days prior to admission a barium enema was attempted but no barium could be made to enter any of the four routes available. Thereafter she had severe sharp pain in the right lower quadrant and groin which radiated up the right side. The discomfort gradually lessened but continued with varying degree.

Physical examination showed no general change from that previously noted. There was marked tenderness and some reddening of the skin in the right lower quadrant. The scar of the more recent operation was slightly edemat-

ous and reddened. Extension of the right leg was painful.

The temperature was 101°, the pulse 110. The respirations were 25.

Examination of the blood showed a red cell count of 5,000,000, with a hemoglobin of 70 per cent. The white cell count was 24,500, 86 per cent polymorphonuclears.

Pain in the right lower quadrant continued and the patient's temperature fluctuated irregularly between 95° and 102°. The cecostomy wound was probed and dilated and about ten ounces of pus released. A catheter was inserted and suction drainage instituted. She improved slightly but on the fourteenth day an abscess of the lower end of the cecostomy wound ruptured spontaneously. Two weeks later material drained from the ileostomy was guaiac-positive. At this time she had a profuse hemorrhage from the cecostomy for which she received a transfusion and parenteral fluids. She improved considerably and the cecostomy catheter drained some pus but no blood. On the forty-sixth day a large, hard, nontender mass was noted filling the entire right side of the abdomen and peculiar glistening grayish granulations appeared in the cecostomy wound. The patient became extremely weak, went rapidly downhill and died on the fifty-first hospital day.

Differential Diagnosis

Dr. Richard H. Sweet: This is a very long history which represents to my mind a characteristic case of ulcerative colitis with all of the symptoms and some of the complications of that disease but it has an unusual termination which I am not able to explain adequately. Why do I call this a typical case of ulcerative colitis? First of all, in reviewing the history we see it begins in a young girl of twelve and we notice at the last admission, that she was twenty-four years old when she died. In other words, this was a long-continued disease with many exacerbations and remissions.

At the first admission we have the characteristic finding of onset of the disease with watery diarrhea, with some fever, some vomiting and then finally the passage of blood, tenesmus, abdominal pain, and all of the symptoms which go with it, an immense loss of weight, and the characteristic finding in the early stages of the disease; namely, through the proctoscope there were seen edema and congestion of the mucous membrane with definite oozing from many small hemorrhagic points in the mucous membrane itself.

On physical examination she was poorly nourished and had the pallor that goes with the disease. We also notice that she had some flexion deformities of the hips and of the knees, probably due to being ill in bed for a considerable period.

Then the third characteristic to note is the stools. She passed stools which often contained a lot of mucus and sometimes large amounts of pus and blood. She had a marked secondary anemia. No ova were found, no evidence of parasites and, I assume, no dysentery organisms. Finally we come to the x-ray finding which is also characteristic; namely, the barium enema showed an outline of the colon with no peristaltic motion, no haustrations. A small contracted smooth colon was outlined by the x-ray.

The patient was treated in the hospital for a considerable period of time, was given transfusions and finally apparently was not doing satisfactorily so they decided to do an ileostomy. Here again we see another characteristic of the disease; namely, the surgeon's description of the colon as he saw it at operation. I think it is an excellent one. He says it was pallid, thickened, and contracted, without normal haustrations. To those who have observed the colon of ulcerative colitis at operation I think that description will be very familiar.

Finally after a long period of time she was discharged and after six months came back for another hospital stay, when an attempt was made to correct the deformities of the extremities but they gave it up because they believed that she was too ill. Then she had a third admission, one month later, at which time she came in with what was apparently a definite acute arthritis with discoloration and swelling about the left knee and left ankle. That is a fairly frequent complication of cases of ulcerative colitis. We notice also that there was some enlargement of the heart and a systolic murmur. Here again we note that she had a significant degree of anemia and that she was running a considerable elevation of temperature which was probably due to arthritis, as nothing is said about the state of her colon at that admission. Then she went home and we hear nothing from her for a period of five years but we notice that when the history was taken that she had not been entirely well during that period but had had remissions and exacerbations of the disease. She came in this time with multiple joint pains and a definite abscess about the knee joint which ruptured spontaneously and drained a purulent discharge. She had other peripheral abscesses. Of course in a very sick patient we not infrequently see peripheral abscesses such as these. It seems to indicate a low resistance to infection. They cultured her stools and rectal mucosa and found hemolytic streptococci. The cultures from the abscesses were negative. It is rather interesting that she was given colonic irrigations with various types of streptococcic antisera with apparently no benefit. Then she went home and stayed away from the hospital for another long period

of time, six and a half years, and then came in with a story which sounds like one of the known complications of ulcerative colitis; namely, acute perforation. We see that two days before this fifth admission, at the age of twenty-four, she had the sudden onset of generalized abdominal pain which was steady in character and therefore suggests that she had a perforation with peritonitis. She had some vomiting but you will notice that whoever took the history must have been thinking of the possibility of obstruction because the note is made that the ileostomy was working well. The assumption therefore would be that there was no element of obstruction. On physical examination the abdomen was distended, with considerable spasm present throughout, more on the right side, in the flank and right costovertebral angle, suggesting that the perforation was in the right half of the colon rather than somewhere else. She had marked temperature elevation. She was obviously quite sick. The white count was 33,700.

Now, of course, they very properly did a laparotomy through a left paramedian incision and found the pelvis full of odorless turbid brownish fluid. In view of the fact that the peritoneum seemed to be free from adhesions, at least my impression from the reading of this description here is that the operator thought there was not a generalized peritonitis because you will notice that he describes the condition of the cecum and ascending colon and then proceeds to make another incision, as if he felt that he could accomplish nothing through the anterior abdominal incision. An incision was, therefore, made in the right flank, I presume with the idea that there was a retroperitoneal perforation from that portion of the bowel which had given a localized abscess in the flank. The region behind the cecum and ascending colon was then explored through that incision and apparently there was no evidence of any perforation or of any necrosis of the intestine at that point.

From that point on I have difficulty in following the course of events because you see the following day a cecostomy was done. I cannot quite see why a cecostomy was done, but a certain amount of pus was evacuated from the bowel and following that the patient seemed to recover and was discharged from the hospital. At the end of two months she came back to the hospital again and this time had definite sepsis in the right half of the right flank region, in the region of the old operative wounds, suggesting a continuation of the sepsis which she had there before. On probing the cecostomy wound about ten ounces of pus was released and then after two more weeks she ruptured some more pus through the lower end of the incision and then finally she had a rather massive hemorrhage from the cecostomy opening which probably came from the large intestine, another one of the complications of ulcerative colitis. Finally after another long stay in the hospital she died after a gradual downhill course, dying, I should assume, from the sepsis that she had resulting from the disease.

It seems to me that it is obvious what the primary disease is but I should be very much interested to find out from the surgeon, if he happens to be here, what the nature of the lesion was in the right lower quadrant which was the apparent cause of her death.

Dr. Tracy B. Mallory: I do not believe the final statement as it appears in the record carries enough force to impress the reader, yet that was the most interesting feature in the case. Perhaps Dr. McKittrick can describe the appearance of that cecostomy wound a little more graphically.

Dr. Leland S. McKittrick: About two or three weeks after her last admission there was still a tube in the opening in the cecum; that is, through the cecostomy wound. Granulation from the first seemed excessive, and soon it blossomed out very rapidly into a fungating mass, five or six centimeters in diameter, with a little opening in the center where the tube went in. That appeared, as I say, probably two to four weeks after her last admission and grew very rapidly in spite of the efforts on the part of the house officers to keep the granulations down.

Dr. Mallory: It is a common thing to see a good deal of edematous swelling and redundancy of the tissues about an enterostomy but this was a much more definite mushroom-like growth than you commonly see under such circumstances. The tissue from the overgrown margin was biopsied and it showed an extremely atypical papillary adenomatous hyperplasia, the cells of which were so undifferentiated that a diagnosis of carcinoma *in situ* was made. There was no evidence of invasion in the specimen which we were given but the cells were so typical that we felt fairly certain of malignancy.

You might tell us what else you know about the case, Dr. McKittrick.

Dr. McKittrick: I should have felt badly if Dr. Sweet had accurately diagnosed this case from the beginning because we changed our minds several times. Our preoperative diagnosis was partly incorrect and partly correct, but mostly incorrect. I thought she had a mesenteric thrombosis secondary to a small bowel obstruction. She had a white cell count of approximately 40,000 at that time. We operated upon her with that diagnosis. She had no peritonitis whatever, a shiny peritoneum throughout, the small bowel absolutely all right, her large bowel in the right side of the abdomen was

about as it is written down here; a large, tense, markedly thickened cecum, with very much injection, and with an area you could see on the mesial aspect of the ascending colon, near the hepatic flexure, which was dusky, but shiny. This was quite a surprise and we concluded that this patient had a constriction of the transverse colon at its beginning, roughly in relation to the hepatic flexure, and that the ileocecal valve, if you will, or at some point between the ileum and the cecum, was also occluded so that she had a blind segment of the colon occluded distally and occluded proximally with reactivation of the disease. This is a complication that we have discussed a good deal but have never seen. Therefore, we went into the right flank hopeful that the cecum was stuck at that area and we could make a hole in it; it was entirely free, however, with about an inch between the abdominal wall and this rigid cecum, and no possible way of getting hold of it without contaminating the entire cavity. Therefore, what we did was to pack three sponges in the hole, one downward to the brim of the pelvis, one upward toward the flank and the other straight in to the cecum, with the idea of producing adhesions. Forty-eight hours later without anesthesia the sponge going directly to the cecum was removed and a trocar cecostomy done, catheter put in, and the trocar slipped out. She was immediately relieved. We wanted her to go home and then come back and have the colon out. She was so well that she refused to come in. So we kept a little tube in the cecum. Then she began to have trouble; the tube had come out and stopped draining and she was readmitted. The second time she never improved. We put her on suction as we did the first time but it did not help. In spite of everything that was done she went downhill and we could not tell why until this lesion cropped out. It at first looked very much like granulation tissue—so much so that the house officers who had seen her every day had been trying to burn it down with silver nitrate. It was firm, however, and nothing else could have looked and felt like that but some form of malignancy. This is the second case of malignant disease that we have had associated with ulcerative colitis in this hospital. The other one was an older patient and the association with ulcerative colitis was not quite so clear cut as this is. Bargen gives an incidence of two and a half per cent in 800 cases.

Clinical Diagnoses

Ulcerative colitis.
Carcinoma of the cecum.

Dr. Richard H. Sweet's Diagnoses

Chronic ulcerative colitis.
Postoperative localized abdominal sepsis.

Anatomic Diagnoses

Ulcerative colitis, chronic.
Carcinomata, multiple, of the ascending colon.
Stenosis, complete, of the ascending colon, the sigmoid and the rectum.
Diverticulum, traction, of the hepatic flexure.
Volvulus of the cecum and the terminal ileum.
Acute ileitis.

Pathologic Discussion

Dr. Mallory: We have a picture which I would like Dr. McKittrick to demonstrate to you.

Dr. McKittrick: This was not quite so satisfactory as it might have been because we were not able to get permission for a complete examination. This had to be drawn without removing anything from the abdomen. Miss McLatchie has done a splendid job, however, in representing the segment of colon as it appeared at postmortem examination. It shows the fungating tumor in the cecum and also the complete constriction of the transverse colon in the upper right hand corner of the picture.

There is one point that was not given in the history and probably is not to be found in the record. She had been followed for a long time in the Out-Patient and was brought into the hospital with the idea of closure of the ileostomy. She had been perfectly well for about five years, was the picture of health, and her disease was not bothering her at all; but someone suggested that she might have the ileostomy closed. At that time it was impossible to do a rectal examination because of complete occlusion of the anal ring. In the Gastrointestinal Clinic we asked for a barium enema. She went to the x-ray department and they could not get a catheter in.

Dr. Mallory: There are several unusual features about the case. You can see in the picture the mass of tissue projecting from the cecostomy wound. The entire cecum and ascending colon were involved in this papillary carcinomatous growth. The transverse colon was white and the mucosa completely atrophic, the rather characteristic late stage of ulcerative colitis. Then sharply at the splenic flexure the colon suddenly disappeared as a hollow viscus but continued as a solid cord, not much over a centimeter, at most 14 mm., in diameter. We made sections across the descending colon at numerous levels and there was no trace of a lumen in any one of them. Microscopic sections showed perfectly good muscular layers with the nerve plexus in between, but the lumen was filled with fat tissue. There was fibrosis but otherwise no remnant of inflammation. It looks exactly like a completely obliterated appendix, but it is quite astonishing that this could occur

all the way from the splenic flexure to the anus. It is no wonder that the attempt at a barium enema was unsuccessful!

There were numerous other complications. She had what appeared to be a traction diverticulum in the region of the hepatic flexure, a very sizable affair, and the cecum had managed to rotate on itself with resulting gangrene of the whole lower end of the cecum and of the terminal ileum.

A PHYSICIAN: Did not the hemorrhage from the bowel have any significance in view of the findings?

DR. MALLORY: I think it did. This loop of ileum which was gangrenous was also adherent to the gangrenous sigmoid, with deep ulceration at that point and the blood which came out of the proximal end of the ileostomy was coming from this gangrenous bit of ileum down here, not from the cecum.

A PHYSICIAN: So it did not necessarily indicate cancerous degeneration?

DR. MALLORY: No.

DR. MCKITTRICK: I saw bloody material coming from the proximal end of the ileostomy, not in relation to the cecum at all, and that was quite confusing.

DR. MALLORY: I will show you a microscopic section of that cecum. It is hardly credible if you do not actually see it.

This is a total cross section of the descending colon. You can see the muscular layers around the outside, the lumen of the gut completely filled with fat and fibrous tissue, occasional spots of lymphocytic infiltration, but essentially the inflammatory process has died out. This is a healed lesion so far as infection is concerned. The tumor from the sigmoid is obviously a papillary carcinoma, polypoid in type. That is the characteristic type that has been seen in similar cases here and elsewhere.

CASE 22322

PRESENTATION OF CASE

An eleven year old white schoolboy was admitted complaining of a tumor of the shoulder.

About two months before entry the patient first complained of pain over the medial aspect of the left clavicle. The pain persisted for about two weeks and then disappeared, but before it had subsided entirely he noticed some swelling in the area involved. The swelling gradually increased until it produced a large oblong mass projecting 3 centimeters above the clavicle. The mass was fixed and caused no discomfort. There was no history of contributory trauma, nor were there any associated symptoms.

Physical examination showed a well developed and nourished boy in no discomfort. He stammered considerably when responding to questions. Over the left clavicle at the junction of the middle and medial thirds there was a firm, nontender, smooth, rounded mass, measuring 4 by 4 centimeters, apparently fixed to the clavicle. No bruit was audible over it. No glands were palpable. The remainder of the physical examination was negative.

The temperature, pulse, and respirations were normal.

Examination of the urine was negative. No Bence-Jones bodies were found. The blood showed a red cell count of 5,050,000, with a hemoglobin of 95 per cent. The white cell count was 9,800, 48 per cent polymorphonuclears. The serum calcium was 10.61 milligrams and the phosphorus 4.4 milligrams. The phosphatase was 5.5 units.

X-ray examination of the cervical spine and chest with special reference to the tumor mass in the left side of the region of the sterno-clavicular joint revealed evidence of multiple congenital anomalies. There was a hemivertebra present in the upper dorsal region on the left associated with considerable deformity of the ribs, the first two ribs on the right being fused. The clavicles are essentially normal. There was no evidence of disease in the bones of the arms, forearms, legs or femora. A tangential view of the sternoclavicular joint revealed a soft tissue mass overlying the left sternoclavicular joint which did not contain calcium or involve the bone.

On the fifteenth hospital day, operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. GRANTLEY W. TAYLOR: In this case we are concerned with a tumor mass of fairly rapid development in an apparently healthy child. Although pain preceded the onset of the tumor, at the time of examination there were no subjective symptoms. Aside from the tumor itself we have nothing but negative evidence on which to base the diagnosis.

The absence of trauma antecedent to the development of the mass justifies us in ruling out a hematoma, a separation of the sternoclavicular joint, or callus formation from a recent clavicular fracture. The likelihood of these injuries is also excluded by the x-ray examination and by the absence of symptoms.

The possibility of some congenital anomaly to explain the mass is raised by the presence of bone anomalies elsewhere, but it is ruled out by the negative x-ray.

Tuberculosis and syphilis are the infections which could give rise to a mass in this region. Tuberculosis could be primary in the cervical spine, or lymph nodes, or in the costal cartilage of the first rib. Gumma in the region of the clavicle is not uncommon and is often mistaken for neoplasm. Record of a Wassermann

or Hinton test would be of interest, although a negative test would not absolutely rule out syphilis. However, in a child there should be other stigmata of congenital syphilis. The character of the masses described does not seem to be consistent with an inflammatory process.

It seems to me we must assume that the mass is a neoplasm, and there is no evidence in the record to suggest that we are dealing with a metastatic tumor, or a generalized process like myeloma or lymphoma. It should be remembered, however, that both myeloma and lymphoma may manifest themselves purely as a localized process, especially in the early stages of the disease.

Assuming a primary localized tumor, the nature of the process must be arrived at on very meager evidence. The brief history suggests fairly rapid growth and makes us suspect malignant disease. The firmness of the mass, presumably without discoloration, and the absence of pulsation or bruit, make hemangioma (and aneurysm) very unlikely possibilities. The absence of x-ray evidence of bone disease practically rules out the possibility of Ewing's sarcoma, osteogenic sarcoma or giant cell tumor of bone.

Thus we are left with a rather rapidly growing firm tumor of the soft parts in this area. We at once think of a fibrosarcoma or a neurofibrosarcoma. Chondroma originating in cartilaginous structures in relation to epiphyseal or articular structures cannot be ruled out. However, this tumor is usually multiple, and usually offers some x-ray evidence of its presence. Giant cell tumor of fascia or periarticular soft parts must be considered a possibility.

I think it is impossible to go farther in the differential diagnosis of this tumor without additional evidence. Such evidence, the most desirable of all, would consist in biopsy. With tumors in certain locations preparations are made for biopsy to be followed at once by radical attack on the tumor mass, if frozen section diagnosis calls for it. Presumably this course was followed in the present case, with a preoperative diagnosis of tumor, probably sarcoma.

Clinical Discussion

Dr. Edward D. Churchill: The x-ray examination showed a definite abnormality of the first rib although it appeared to be a congenital bifurcation of this rib rather than a tumor. On examination the tumor appeared to arise from the clavicle but no x-ray abnormality of the clavicle was demonstrable.

This case represents a classical picture in surgical pathology but one that the present generation sees so rarely that the correct diagnosis was not made. A familiar examination question in surgery for years has been the differential diagnosis of tumors arising in the inner third of the clavicle, and gumma ranks high in the differential diagnosis. The incidence of untreated congenital lues is now so small, at least in our clinic, that although this diagnosis was considered, the alternative diagnosis of tumor was deemed more likely. Through an error the Wassermann report was recorded as negative before operation, so we proceeded along the lines that Dr. Taylor has indicated to do a biopsy of the tumor. The simplest form of biopsy seemed to be a complete extirpation, and this was carried out. A transverse incision was made and the mass was found closely fixed to the inner end of the clavicle in the region of the origin of the sternomastoid muscle. This muscle and others surrounding it were involved in an infiltrating and sclerosing proliferation of fibrous tissue. The periosteum was separated from the clavicle beneath the tumor and stripped from the bone revealing pitted, atrophic bone. The mass was removed in its entirety. Healing was accomplished by first intention. The peculiarities of the "tumor" on dissection led us to do this conservative operation rather than embark upon a radical resection of the clavicle which in the light of the pathological report would have been inadvisable. When the pathologist reported a gumma we were, of course, considerably chagrined that the diagnosis had not been made. A check-up on the serological reports showed a weakly positive Wassermann and a positive Hinton. On removing the boy's spectacles, facies were exposed that were in themselves diagnostic of congenital lues. The incisor teeth were suggestive and the six-year molars were missing from early decay. Fortunately our surgical error was not a serious one but I feel distinctly that it was one that would not have been made by the surgical service twenty-five years ago.

Preoperative Diagnosis

Tumor in the region of the left clavicle.

Dr. Grantley W. Taylor's Diagnosis

Tumor of the left clavicle, probably sarcoma.

Pathologic Diagnosis

Gumma in the region of left clavicle.

Pathologic Discussion

Dr. Tracy B. Mallory: This case has been presented to Dr. Taylor on the basis of the evidence which was at hand at the time of the patient's operation. As Dr. Taylor pointed out, a definite diagnosis in such cases is more often than not impossible without the aid of biopsy and microscopic examination, and on this

basis the child was operated on without waiting for more elaborate diagnostic measures.

When the tumor was cut down upon, it proved to be a mass of dense fibrous tissue in the center of which was an area of yellowish necrosis. A frozen section was strongly suggestive of a gumma and this diagnosis was eventually confirmed. It was felt that excision was nevertheless the most suitable form of treatment and the entire mass, 8 x 5 x 4 cm. in size, was dissected out.

As Dr. Churchill has pointed out, an untreated congenital syphilitic has become a great rarity in this clinic and very few of our generation of physicians have seen a significant number of cases. An exception, however, is Dr. Cheever, who for years has been particularly interested in the problem of congenital syphilis and when he was called in consultation to see this patient he pointed out the, to him, obvious stigmata which Dr. Churchill has mentioned.

This is the type of case in which to my mind the Hinton test is most valuable. It is considerably more sensitive than the standard Wassermann reaction and, as is well known, will pick up a great many cases of treated syphilis which the Wassermann test misses. Such cases, however, are rarely particularly important unless the history has been falsified. In congenital syphilis, however, it is not infrequent to find a definitely positive Hinton test in the face of an entirely negative Wassermann reaction. I was particularly impressed a few years ago in checking over the results of the Hinton test to discover two children with positive Hinton tests, negative Wassermann reactions, and no stigmata whatever of congenital syphilis. Nevertheless both children belonged to large families in which older siblings had had definite stigmata, and positive Wassermann reactions, and in each family the first child had been stillborn.

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THE TWO HUNDREDTH ANNIVERSARY OF THE NOTABLE CONTRIBUTION ON SCARLET FEVER BY WILLIAM DOUGLASS[1]

WHILE Sydenham in 1676 was the first to give us the name "scarlet fever" (Febris Scarlatina), it is somewhat doubtful whether he described the disease to which this name has since been given. Indeed, it is not impossible, as Weaver[2] has suggested that Sydenham was describing German measles, because no mention was made of the throat or tongue, and, furthermore, it was of such a mild character as to be a "disease in name only".[3]

It remained for William Douglass of Boston to be the first to describe scarlet fever in unmistakable terms. In 1736 he wrote a treatise on the epidemic of this disease which started in 1735 in Kingston, New Hampshire, and spread in a severe form over New England. Douglass was the first to describe the course of an epidemic of this disease and give a clinical description of the mild and severe forms. While we certainly cannot go so far as Weaver, who affirms that the Douglass treatise was "the first real contribution of America to the medical literature of the world," it was undoubtedly the second,[4] and a true landmark in our knowledge of scarlet fever. It is really of greater importance than Sydenham's term "Scarlet fever," and serves as the forerunner of Withering's[5] classic in 1779.

Zabdiel Boylston's[6] report on smallpox inoculation is certainly the first great American contribution to medical literature. Boylston's practice of inoculation was instigated by Cotton Mather, who had become interested in the possibilities of this procedure at least five years before and had more recently borrowed a treatise on the subject from Douglass.[7] The Boylston report exerted a profound influence in London. The publication of Douglass ten years later was of an entirely different order, consisting of brilliant original observations based on a scholarly foundation. Unfortunately, Douglass is remembered for his vehement opposition to inoculation against smallpox and for his abusive attacks on Boylston,[8] and more particularly on Cotton Mather.[9] From the viewpoint of the history of smallpox, Douglass is a sinister figure, who in an offensive manner impeded the progress of inoculation, but as seen through the rose-colored spectacles of a recognized authority on scarlet fever, Weaver,[10] he is made out to be a man with faults, it is true, but a physician of learning and ability, a botanist and historian as well as a public-spirited citizen. Weaver reminds us that Cotton Mather was early in his career an ardent persecutor of witches, a point harped on unmercifully by Douglass. We must bear in mind that Douglass genuinely disapproved of Mather's instigation of the new method, and, like the majority of doctors, resented the entry of the church into the domain of medicine, just as Sydenham was at first opposed to the use of the Jesuits' Bark from Peru in cases of malaria. In 1730 and again in 1751 Douglass wrote in favor of inoculation, yet it was not until 1754 that the College of Physicians of London ventured to give it support. At this 200th anniversary of his famous treatise on scarlet fever we get some satisfaction in the realization that at the time of its publication Douglass had withdrawn his opposition to inoculation, even though he would never give due credit to Mather and Boylston.

As the only physician in Boston at that time who held a medical degree[11] Douglass was probably the leading consultant of the city. He openly denounced the "kidnapping" of seamen on the ships in Boston Harbor for service in the Royal Navy, and objected with clear reasoning to the dangers of inflation with paper Colonial currency.[12] The township of Douglass in Massa-

chusetts was named after him in 1746.[12] However, his name is indelibly associated with the disease he described and to which he gave too long a name.[13] He remarked that the brilliant forms of it "were called by some a scarlet fever". The quaint terms and expressions of the period sometimes leave the modern reader in some doubt as to what was meant, but it is highly probable that the readers of medicine two hundred years from now will have much greater difficulty understanding the present-day immunologic jargon applied to the streptococcus. In 1825 the Douglass treatise was reprinted in full in *The New England Journal of Medicine and Surgery* as a masterpiece of which New England might well be proud.

* * * * *

Sydenham's subtle remark that scarlet fever is a disease in name only might well be applied today to this malady, the problems of which rock the conclaves of the medical profession at home and abroad. No less than two hundred strains of hemolytic streptococci divided into four groups have been found capable of producing a scarlet fever toxin. Bacteriologists and clinicians are divided among themselves on the very question of the entity of the disease, its cause, nature, prevention, and treatment. That a single strain of streptococcus can produce in one individual scarlet fever, in another sore throat without a rash, and in still another puerperal fever, has long been suspected by some and denied by others. Whether the rash is or is not an allergic manifestation; whether the toxoid is a true toxoid and as efficacious as the toxin; and, finally, the relative merits of antitoxin and convalescent serum, are all matters under discussion. And last but not least we have the Dick Patent—a sort of Prohibition Amendment to the Hippocratic Oath—which we like to think would outrage the ethics of Sydenham, Withering, and even William Douglass.

REFERENCES

1. Douglass, William: The Practical History of a New Epidemical Eruptive Miliary Fever, with an Angina Ulcusculosa, which prevailed in New England in the Years 1735 and 1736. (Boston, 1736.) Reprinted in New Eng. J. Med. and Surg., 14, (1825), 1.
2. Weaver, George H.: Scarlet fever. Abt's Pediat. **6**:298, 299, 1925.
3. Sydenham, Thomae: Opera Omnia. Edited by Greenhill, G. A. London, 1844, p. 244.
4. Bartlett, Josiah: A Dissertation on the Progress of Medical Science in the Commonwealth of Massachusetts. (Read before the Mass. Med. Soc., June 6, 1810.) Boston, 1810, p. 7.
5. Withering, William: An Account of the Scarlet Fever and Sore Throat or Scarlatina Angiosa. London, 1779.
6. Boylston, Zabdiel: An Historical *Account* of the *Small-Pox Inoculated* in *New England*, Upon all Sorts of Persons, *Whites*, *Blacks* and of all Ages and Constitutions, With Some Account of the Nature of the Infection in the NATURAL and INOCULATED Way, and their different effects on HUMAN BODIES. With some *Short Directions* to the *Unexperienced* in this Method of Practice. Humbly dedicated to her Royal Highness the Princess of Wales by *Zabdiel* BOYLSTON, Physician. London, 1726.
7. Kittredge, George L.: Some Lost Works of Cotton Mather. Cambridge, 1912 (Mass. Hist. Soc., 45), pp. 423-427.
8. Fitz, Reginald H.: Zabdiel Boylston, inoculator, and the epidemic of smallpox in Boston in 1721. Bull. Johns Hopkins Hosp. **22**:317, 1911.
9. Kittredge, George L.: Mather's Several Reasons. Cleveland, 1921, p. 12.
10. Weaver, George H.: Life and Writings of William Douglass, M.D., 1691-1752. Bull. Soc. M. History, Chicago, 2, 4, 1921, p. 205.
11. Douglass is said to have been a pupil of Pitcairn, at one time Professor of Medicine at Leyden. From this irascible Scotchman, Douglass may well have acquired his disrespect for the church. It is not known where Douglass took his degree, but he was one of a group of highly educated Scotch physicians who came to America in the early part of the eighteenth century. Douglass was born in Gifford, County of Haddington, Scotland, about 1691, and arrived in Boston in 1716. He died in 1752. (Weaver, 1921, pp. 230-231.)
12. Weaver, 1921, p. 244.
13. Apparently the autopsies which he mentions having performed in some of these cases were the first recorded in America. He described the findings also in a letter to Colden, who in turn transmitted them to Fothergill. (Weaver, 1921, p. 240.)

APPRAISAL OF VITAMIN D MILKS

An impartial review, which should be of general value, has been made by Philip C. Jeans of Iowa City to determine the relative values of different varieties of vitamin D milk for infants. (*J. A. M. A.* **106:** 2066 and 2150, June 13 and 20, 1936.) This review consists of an interpretation of all the important recent literature on the subject. The commercial vitamin D milks include, broadly, milk directly irradiated, milk to which has been added a cod liver oil concentrate (Zucker process) and milk from cows whose ration includes an irradiated vegetable source of vitamin D, such as "yeast milk".

There are apparently two, and possibly only two, varieties of vitamin D—that of animal origin and that of vegetable origin, and it is probable that the animal does not change the biologic form of the vitamin when it makes the vitamin D available in the milk. While all animal sources may have a vitamin D of the same value, as may all vegetable sources, vitamin D of animal source appears to be more potent than vitamin D of vegetable source, possibly in the ratio of 1.5:1 when vitamin D milks are compared.

The information available concerning the value of animal source vitamin appears to be greater than that concerning vegetable source vitamin. Thus it is known that animal source vitamin D milk containing 135 U. S. P. rat units to the quart will prevent rickets, but this amount of vitamin D approaches closely the minimum effective level. Less vitamin D may initiate the healing of rickets than is necessary consistently to prevent it.

Moreover, the amount of vitamin D that barely prevents rickets does not permit the best growth of infants, nor does it permit retention of calcium and phosphorus as great as those considered desirable. An adequate amount of animal source vitamin D is considered to be the amount present in one teaspoonful of average high grade cod liver oil, or in milk containing 400 units to the quart. The same information about vegetable source vitamin has not been determined, nor is the minimal adequate dosage for either type as yet known.

THIS WEEK'S ISSUE

CONTAINS articles by the following-named authors:

WEILLE, FRANCIS L. B.S., M.D. Harvard University Medical School 1925. Assistant Surgeon, Massachusetts Eye and Ear Infirmary. Assistant in Otolaryngology, Harvard University Medical School. His subject is Studies in Asthma. XIX. The Nose and Throat in Five Hundred Cases of Asthma. Page 235. Address: 395 Commonwealth Avenue, Boston, Mass.

JOHNSON, ALLEN S. A.B., B.A., M.D. Harvard University Medical School 1927. F.A.C.P. Visiting Physician, Tuberculosis Division, Springfield Health Department Hospital. Assistant Visiting Physician, Springfield Hospital. His subject is Factors Influencing the Development of Tuberculous Infection in Childhood. Page 239. Address: 121 Chestnut Street, Springfield, Mass.

CHEEVER, AUSTIN W. A.B., M.D. Harvard University Medical School 1914. Assistant, Department of Dermatology and Syphilology, Harvard University Medical School. Assistant Physician to Syphilis Out-Patient Department, Massachusetts General Hospital. Visiting Dermatologist, Beth Israel Hospital. Assistant Dermatologist, Children's Hospital. Consulting Dermatologist, Waltham Hospital; Brockton Hospital; Goddard Hospital, Brockton; Framingham Hospital and Florence Crittenton Home. His subject is Progress in the Treatment and Diagnosis of Syphilis, 1935. Page 242. Address: 41 Bay State Road, Boston, Mass.

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1935	Dean, Stanley Rochelle, Taunton	38
1935	Derow, Matthew Arnold, Boston	10
1936	DeShon, Herbert Jackson, Boston	30
1935	Diamond, Louis Klein, Boston	11
1936	Ditmore, David Claude, Centre Newton	32
1936	Donaghy, George Everett, Dorchester	12
1936	Donahue, Lewis Charles, Needham	12
1936	*Dorfman, William Wolf, Revere	20
1936	Downing, Francis Harold, Boston	35
1936	Doyle, Joseph Bernard, Chestnut Hill	11
1935	†Duff, Paul Harrington, Peabody	11
1935	Dutton, Robert, Wakefield	11
1935	East, Harry Colin, Andover	12
1935	Eastwood, Medora Viola, Plymouth	12
1935	Edgar, William Ladell, Athol	8
1935	d'Elseaux, Frank Christian, Newton	11
1935	Eversole, Urban Harris, Boston	49
1936	Farnsworth, Dana Lyda, Williamstown	11
1935	†Faxon, Nathaniel Wales, Chestnut Hill	11
1936	Federkiewicz, John Joseph, Dorchester	12
1936	Ferriter, Thomas Francis, Lowell	12
1936	Fiedler, Howard Taft, Westborough	8
1936	Fieldman, Jonah, Brockton	12
1935	Finesinger, Jacob Ellis, Cambridge	18
1935	Fleming, Elizabeth Putnam, Beverly	33
1936	Foley, John Thomas, Jr., South Boston	12
1935	Fortin, Philip Frederick, New Bedford	46
1936	Freedman, Jacob Phillip, Dorchester	12
1936	Freeman, Norman Easton, Cambridge	1
1935	*Freitag, Abraham, Lynn	50
1935	*Fuller, Wilfred Joy, South Yarmouth	6
1935	Gaebler, William Charles, Foxborough	31
1935	Gardiner, Harry Miller, Westborough	8
1935	Gates, Olive, Newton Centre	1
1936	*Gatsopoulos, John Konstas, Lowell	36
1935	Gill, Charles Edward, Westfield	30
1936	Gillis, Edwin James, Westfield	12
1935	Gingras, Albert Francis, Medford	12

1935 *Gittleman, Irving, Dorchester 44
1935 Glick, Harry Sumner, Dorchester 12
1936 Goodnow, Chester Lloyd, Franklin 12
1936 Green, William Richard, Lowell 12
1936 Greene, Jeremiah Evarts, Newton Centre 11
1935 Gurry, Joseph Francis, Lawrence 19
1935 Graybiel, Ashton, Cambridge 11
1935 Haigis, Peter, Foxborough 10
1936 Hajjar, Solomon Gantous, Lowell 43
1935 Haling, Raymond Francis, Southbridge 10
1936 Hall, Leonard John, Lowell 12
1936 Ham, Thomas Hale, Boston 33
1936 Hammer, Joseph, Wellesley 10
1935 Harrington, Harold Francis, Seekonk 12
1936 Harwood, Reed, Brookline 11
1935 Hathaway, John Seabury, Cambridge 11
1936 Hayes, Paul Thomas, Worcester 12
1936 Healy, John Francis, Lowell 12
1935 Heller, Hyman, Webster 15
1935 Hennessey, James Alfred, Brookline 12
1935 Higgins, Donald Ellwood, Cotuit 11
1936 Hill, William Robinson, Taunton 11
1935 Hindman, James Harold, Lawrence 12
1935 *Hoefer, Paul Frederick Adam, Boston 37
1935 Holt, William Leland, Jr., Brookline 11
1935 Hook, William Gilbert, Danvers 10
1936 Hooker, Sanford Burton, West Roxbury 10
1935 Hosman, Israel Daniel, Chicopee 40
1936 Hosmer, Miriam Jewell, Wilmington 38
1935 Howard, Edgerton McClellan, Boston 29
1935 Hoyt, William Fenn, Longmeadow 11
1936 Ives, Lionel Mortimer, Westwood 41
1935 Johnson, Raymond Edwin, Newton Highlands 11
1936 Kahn, Lester Sydney, Brookline 12
1935 Kelleher, John Joseph, Jr., Lawrence 12
1935 Kelley, John Samuel, Jamaica Plain 10
1936 Kelley, William Joseph, Roxbury 12
1935 Kenler, Maurice David, New Bedford 12
1936 King, Samuel Joshua, Boston 11
1936 Koretsky, Leo, Chelsea 21
1936 Kranes, Alfred, Boston 11
1935 *Krobalski, Joseph, Lynn 20
1936 Krumbhaar, George Douglas, Brookline 11
1936 Lavner, Gerald, Haverhill 2
1935 Ledger, George H., Taunton 46
1936 Leone, Joseph Peter, Quincy 5
1936 Levine, Louis, Cambridge 12
1936 Lipton, Joseph Harold, Quincy 12
1935 *Lobo, Jose Paulo, Fall River 51
1935 Loizeaux, Marion Cotton, Wellesley 33
1936 Low, Merritt Burnham, Roxbury 11
1935 Lynch, George William, Woburn 11
1935 MacKinnon, George Lincoln, Wollaston 12
1935 Maguire, James Alfred, Atlantic 12
1936 Mahoney, Alfred Vincent, Quincy 12
1935 Mahoney, Patrick James, Brookline 11
1935 Mallory, George Kenneth, Brookline 11
1935 Maloney, John David, Waverley 12
1935 Manwell, Claire Cutten, Northampton 5
1935 Manwell, Edward Jones, Northampton 5
1936 Marshall, Samuel Frederick, Newton 18
1936 Mayo, Leroy Edward, Worcester 12
1935 McAdoo, Hosea Webster, Arlington 27
1936 McGinn, Sylvester, Brookline 11
1936 *McHugh, William Patten, Peabody 6
1935 McLean, Sterling Alexander, Middleborough 9
1936 Mendenhall, Walter Leslie, Cambridge 42
1936 Merrick, Francis Humphrey, Dorchester 10
1935 Merritt, Hiram Houston, Cambridge 18
1936 *Messer, Samuel Irving, Waltham 44
1936 Montag, Paul Peter, Worcester 12
1935 Moore, Carlton William, Georgetown 10
1935 Moore, Merrill, Boston 3
1936 Mower, Etamar Anses, Cambridge 26
1935 Moxness, Bennie Arthur, Northampton 19
1936 *Murphy, Lawrence, Newburyport 45
1936 Murray, Thomas Oscar, Worcester 12
1935 Nason, Louis Howard, Boston 11
1935 Naterman, Hyman Louis, Roxbury 12
1936 Nathanson, Seymour Isaac, Fitchburg 7
1935 Newell, Howard Winthrop, Westfield 9
1936 Nicgorski, Eugene John, Gardner 10
1936 Nichols, Howard Gage, Haverhill 11
1936 Norton, Paul Lawrence, Cambridge 11
1935 O'Neill, Hugh Wilson, Jamaica Plain 4
1935 *Orlansky, George J., Dorchester 20
1936 *Oskar, Paul Augustine, Lawrence 6
1936 Palmer, Edwin James, East Gardner 22
1935 Pappas, James Peter, Cambridge 12
1935 Parker, Frederic, Jr., Newton Centre 11
1936 Pearson, Grosvenor Benjamin, Roxbury 34
1936 Peatick, George E., Westborough 8
1935 *Peters, Francis Donald, Somerville 6
1936 Pfeifer, Ladislaw Eugene, Springfield 12
1935 *Pipkin, Hubert Alexander, Pittsfield 6
1936 Poirier, Theophane Marcel, Marlborough 19
1936 *Pratt, Ernest F., Lowell 6
1935 Prien, Edwin Louis, Boston 11
1936 Rabnowitz, Henry, Springfield 38
1936 Ramsdell, Edward Snyder, Worcester 12
1936 Reinherz, George, Chelsea 12
1935 Ring, Lina Barbara Taylor, Arlington 10
1936 Riseman, Benjamin, Dorchester 12
1935 Robbins, Eleanor, Holliston 52
1935 Roberts, Charles Dewees, Milton 11
1936 Roberts, Helen Lucile, Cambridge 38
1935 Robinson, Hortensia Amanda Farrall, Newton Centre 18
1936 Robinson, Leon Jerome, Palmer 10
1936 Roche, William David, Worcester 12
1935 Rothschild, David, Foxborough 9
1935 Rovner, Miah Henry, Norfolk 12
1936 Russman, Reuben, Belmont 12
1935 Russo, Anthony Reginald, Somerville 10
1935 *Salemi, Charles, Boston 6
1935 Scelso, Salvatore, Medford 10
1935 Schneider, Benjamin, Monson 12
1935 *Schraffa, Louis Emilio, East Boston 6
1935 Sewall, Weston, Roslindale 11
1936 Shannon, Edward Terrence, Fall River 19
1936 Sharp, Morris Louis, Foxborough 12
1935 Shea, Philip Joseph, North Cambridge 19
1936 *Siegel, Max, Mattapan 45
1935 Simmons, Fred Albert, Jr., Boston 11
1936 Simon, Benjamin, Worcester 23
1936 Sloane, Albert Edward, Chelsea 12
1936 Smedal, Magnus Ingvald, Brookline 11
1935 Smith, Herbert Harold, Brookline 10
1936 Spritz, Benjamin, Chelsea 12
1935 Staples, Clarke, Chestnut Hill 11
1936 Stella, Frank, Wakefield 12
1935 Strieder, John William, Arlington 11
1935 Talkington, Perry Clement, Taunton 53
1936 Tarnower, Samuel M., Pittsfield 31
1935 *Tavares, John Mathias, Somerville 54
1935 Taylor, Zella Eileen, Winchester 10
1935 Thompson, Milton Strong, Jr., Boston 11
1936 Thompson, William Graham, Andover 11
1936 Tompkins, Mary Isabel, Boston 12
1936 Trapp, Carl Edward, Dorchester 10
1936 Trevett, Laurence Davies, Stockbridge 18
1936 Tribeman, Melvin Samuel, Haverhill 12
1935 Turner, Ralph Gordon, Boston 15
1935 Tynan, James Joseph, East Milton 30
1935 *Ullian, Hyman Bertram, Everett 6
1936 Valliere, Leon J., Lowell 9
1936 Vernon, Hollis Edward, Spencer 9
1936 *Vidoli, Marius Francis, Newton Highlands 47
1935 Ward, Arthur Henry, Winchendon 12
1936 Ward, John Langdon, Medford 11
1936 Warren, Harry Allen, Boston 25
1935 Welch, Robert Francis, Brockton 11

1935	Wilcox, Paul Harlan, East Gardner	38
1936	Williams, Walter Wilkinson, Springfield	13
1936	Wolanske, Stephen, Worcester	12
1935	Younge, Paul Adolph, Milton	11
1935	Zovickian, Hovhannes, Watertown	11

*Indicates that the candidate's diploma was approved by the standing Committee on Medical Education and Medical Diplomas, he was given a personal interview by this committee and permitted to take an examination before a Board of Censors.

†Readmitted by Censors.

Total = 248

KEY TO MEDICAL COLLEGES

1. Yale University School of Medicine
2. Syracuse University College of Medicine
3. Vanderbilt University School of Medicine
4. University of Pennsylvania School of Medicine
5. University of Rochester School of Medicine
6. Middlesex College of Medicine and Surgery
7. University of Vermont College of Medicine
8. Hahnemann Medical College and Hospital of Philadelphia
9. McGill University Faculty of Medicine
10. Boston University School of Medicine
11. Harvard University Medical School
12. Tufts College Medical School
13. Albany Medical College
14. University of Vienna Medical School
15. Rush Medical College of the University of Chicago
16. University of Louisville School of Medicine
17. State University of Iowa College of Medicine
18. Johns Hopkins University School of Medicine
19. Georgetown University School of Medicine
20. College of Physicians and Surgeons, Boston
21. Jefferson Medical College of Philadelphia
22. Medical College of Virginia
23. Washington University School of Medicine
24. University of California Medical School
25. Northwestern University Medical School
26. Laval University Faculty of Medicine
27. Tulane University of Louisiana School of Medicine
28. College of Medical Evangelists
29. Columbia University, College of Physicians and Surgeons
30. University of Maryland School of Medicine and the College of Physicians and Surgeons
31. New York Homeopathic Medical College and Flower Hospital
32. University of Minnesota Medical School
33. Cornell University Medical College
34. University of Pittsburgh School of Medicine
35. Stanford University School of Medicine
36. National University of Athens School of Medicine, Greece
37. University of Berlin
38. University of Michigan Medical School
39. Long Island College Hospital
40. University of Chicago School of Medicine
41. University of Toronto Faculty of Medicine
42. Drake University College of Medicine
43. St. Louis University School of Medicine
44. Missouri College of Medicine and Science
45. St. Louis College of Physicians and Surgeons
46. Temple University School of Medicine
47. Royal Universitii di Roma, Italy
48. Royal Frederic University, Tonsberg, Norway
49. University of Kansas School of Medicine
50. University of Pecs, Hungary
51. Medico-Chirurgical School of Lisbon
52. Woman's Medical College of Pennsylvania
53. Baylor University College of Medicine
54. Faculdade de Medicina e Cirurgia da Universidade de Coimbra, Portugal

MISCELLANY

THE CAPE COD HOSPITAL APPEALS FOR FUNDS

The Cape Cod Hospital located in Hyannis which is the only general hospital serving Barnstable County is appealing for funds to meet a deficit.

This hospital was established in 1919 and has grown so that now there are eighty beds including fifteen bassinets. The service is supplied to a thirty-thousand year-round population augmented by about one hundred thousand summer residents. The American College of Surgeons has given this hospital a Class A rating.

This is an active hospital with an average of 52.11 patients per day.

The equipment is up-to-date and the management excellent.

The President, Charles L. Ayling, has sent out 4,000 letters setting forth the needs of the hospital.

HEALTH OFFICERS' MONTHLY STATEMENT OF VENEREAL DISEASES REPORTED IN THE NEW ENGLAND STATES

MAY, 1936

This statement is issued monthly for the information of health officers in order to furnish current data as to the prevalence of the venereal diseases. The following reports were received from State Health Officers. The figures are preliminary and subject to correction. It is hoped that this will stimulate more complete reporting of these diseases.

State	Syphilis		Gonorrhea	
	Cases reported during month	Monthly case rates per 10,000 population	Cases reported during month	Monthly case rates per 10,000 population
Connecticut	240	1.45	96	.58
Maine	53	.66	42	.52
Massachusetts	446	1.03	441	1.02
New Hampshire	15	.32	16	.34
Rhode Island	112	1.59	46	.65
Vermont	21	.58	21	.58

Treasury Department—*U. S. Public Health Service.*

EDITORIAL NOTE

Figures for the whole United States show that there were reported in May, 1936, 22,959 cases of syphilis and 12,442 cases of gonorrhea.

These show the great public health problem in dealing with venereal diseases.

ILLUSTRIOUS CONTRIBUTORS TO PUBLIC HEALTH

At the time of the dedication exercises of the New York City building to house the Department of Health, Hospitals and Sanitation and the office of

the Chief Medical Examiner, November 26, 1936, Dr. Charles Frederick Bolduan prepared a sketch of the building with the names of twenty-nine Contributors to Public Health carved on the frieze.

This sketch was distributed to the guests present on that occasion and in response to requests was put in permanent form in a brochure of thirty-three pages made possible through the generosity of Mr. Lucius N. Littauer.

The selection of names appearing on the frieze was made by a small committee appointed by the Public Health Committee of the New York Academy of Medicine. Dr. Samuel W. Lambert was chairman of this small committee. It was an arduous task to consider the great number of those whose names are identified with public health, especially since the space permitted only the limited number designated above.

In the brochure, in addition to the portraits of the selected persons, Dr. Bolduan makes a brief reference to the distinctive contribution of each. The distribution of the names on the frieze is shown herewith:

Worth Street (South Façade)	Lafayette Street (West Façade)
Moses	Koch
Jenner	Behring
Ramazzini	Pasteur
Hippocrates	Leeuwenhoek
Paracelsus	Ehrlich
Pinel	Billings
Lind	Harvey
Centre Street (East Façade)	Leonard Street (North Façade)
Farr	Bard
Howard	Semmelweis
Lister	Welch
Nightingale	Stephen Smith
Shattuck	E. B. Dalton
Morton	Biggs
Marion Sims	

Court Facing Leonard Street

Walter Reed
Gorgas

In closing the brief biographical sketches of these selected persons, Dr. Bolduan pays especial tribute to Dr. Hermann M. Biggs in these words: "We have left for the last the name of Hermann M. Biggs. He, as the moving spirit of the New York City Department of Health from the late 80's until well into our present century, was quick to seize upon all the contributions of bacteriology and the other medical sciences and apply them to improving the health of our city. His vision brought about the establishment in this city of the first municipal bacteriological diagnostic laboratory in the world, also the first antitoxin laboratory outside of Europe. It was his understanding of the medical and social problems, and his organizing ability which made New York City the pioneer in tuberculosis control measures. It was he who first attacked the venereal diseases as a public health problem, and established the first public laboratory for making Wassermann blood tests free of charge. Biggs was the outstanding pioneer in bringing to the public the fruits of the great discoveries mentioned above — and bringing them promptly and effectively. He was not a discoverer, but a typical man of action; an accelerator and catalyzer. Without him, public health would have lagged far behind the brilliant strides made by research workers. With him, we have kept pace as we should."

The building and the activities of this great center of progress in public health work will justify a pilgrimage to New York even from the far-away cities of the country.

RESULTS OF TUBERCULIN TESTING BY COUNTIES FOR THE SCHOOL YEAR 1934-1935

GRADES 7, 9 AND 11—PUBLIC AND PAROCHIAL SCHOOLS

County	No. Tested	Per Cent Reactors
Barnstable	667	17
Berkshire	2,456	17
Bristol	333	37
Dukes	82	28
Essex	8,802	24
Franklin	2,294	12
Hampden	8,671	19
Hampshire	363	31
Middlesex	13,585	23
Nantucket	62	8
Norfolk	2,977	27
Plymouth	93	21
Suffolk (See note)	10,124	16
Worcester	8,248	23

NOTE: Boston not included—no testing done in schools. Figures for Winthrop and Chelsea are for school year 1933-1934—no testing done school year 1934-1935.—*Submitted by the Massachusetts Tuberculosis League.*

THE APPROPRIATIONS OF THE ROCKEFELLER FOUNDATION

According to an annual report just published, The Rockefeller Foundation in 1935 expended $12,725,439.

Of especial interest to medicine are the following allocations:

PUBLIC HEALTH

The International Health Division of The Rockefeller Foundation in 1935 operated on a budget of $2,200,000. Grants were made for yellow fever studies in Brazil; for research on yellow fever, malaria, and other diseases at the laboratories of the International Health Division at the Rockefeller Institute; for field research on malaria in Cuba, Puerto Rico, Albania, Bulgaria, Greece, Italy, Portugal, Spain, and India, and for laboratory studies of this disease at the University of Chicago; for

demonstrations in the control of malaria in Colombia, Nicaragua, Salvador, and Albania; for investigations and control of hookworm disease in Egypt, and for surveys to determine the present status of hookworm disease in areas of North Carolina where campaigns for its control were carried on from 1910 to 1915 by the Rockefeller Sanitary Commission and the International Health Commission; for studies of tuberculosis at Cornell University Medical College, and in Alabama, Austria, Jamaica, and Tennessee; for the study of yaws in Jamaica and of undulant fever in France; for research at Columbia University on the common cold; for diphtheria investigations in Austria and Peiping, China; for studies on influenza, to be carried out for the most part in the United States; for research on typhoid fever at the State Institute and School of Hygiene, Warsaw, Poland; for studies of mental hygiene in Tennessee and in Baltimore, Maryland; for the development, under the Polish National Department of Health, of a division of mental hygiene in the Warsaw School of Hygiene; and for research on smallpox vaccine at the National Institute of Hygiene in Madrid, Spain.

In addition, local and central government health services were aided in many states and countries; and public health education was furthered by grants to institutions and by the maintenance of a fellowship program providing advanced training in public health and public health nursing for 188 persons.

MEDICAL SCIENCES

During 1935 The Rockefeller Foundation appropriated $2,733,050 for work in the field of medical science. Of this sum, $1,459,450 was contributed to projects for the advancement of psychiatry.

For the development of teaching and research in psychiatry grants were made to the Institute for Psychoanalysis, Chicago; The Johns Hopkins University School of Medicine, for teaching in child psychiatry; the Institute of the Pennsylvania Hospital, for training in psychiatry; the Harvard Medical School and the Massachusetts General Hospital, for a joint program in psychiatric training; the University of Michigan Medical School, to extend the scope of psychiatric training; the School of Medicine of the University of Colorado, for the teaching of psychiatry in medical, surgical, and obstetrical clinics; and to the University of Chicago School of Medicine, to establish and maintain a subdepartment of psychiatry.

A contribution toward a laboratory building was given to the National Hospital, Queen Square, London.

Fourteen research undertakings in clinical psychiatry and related fields received grants ranging in duration from one year to five years, and in amount from $5,000 to $90,000. The institutions and fields of work thus aided were: Maudsley Hospital, London, research in psychiatry; University of London, Galton Laboratory, studies in human genetics in relation to mental disease; Columbia University, College of Physicians and Surgeons, psychiatric research; University of Amsterdam and the Worcester State Hospital, Massachusetts, research on dementia praecox; Cornell University Medical College, studies on reflex behavior; University of Chicago, study of the physiology of sleep; Northwestern University Medical School, research in neuroanatomy; New York University College of Medicine, research in experimental neurology; Dartmouth College Medical School, research in physiological optics; University of Pennsylvania School of Medicine, research on growth of living tissues; Harvard Infantile Paralysis Commission, field studies in North Carolina; Chicago Area Project, field study of abnormal behavior, and the Institute of the Educational Sciences, Geneva, Switzerland, research in child psychology.

In connection with the application of modern psychiatric knowledge, grants were made to the National Committee for Mental Hygiene, in New York City, the North Carolina Commission for the Study of the Care of the Insane and Mental Defectives, and the Massachusetts Department of Mental Diseases.

During the year the Foundation administered seventy-two fellowships in the medical sciences.

Other important appropriations are the following: Natural Sciences, $2,426,125; Social Sciences, $3,807,500; Humanities, $1,169,440 and large grants to the Chinese Mass Education Movement.

CHINA PROGRAM

The Chinese Mass Education Movement, the Colleges of Public Affairs and Natural Sciences at Yenching University, and the Institute of Economics of Nankai University received aid from the Foundation during 1935. In the part of the China Program centering around Nanking in South China, support was extended to the National Health Administration, to the Commission on Medical Education, and to three institutions interested in agricultural development: the University of Nanking, for its departments of agricultural economics and science; the National Central University, for work in animal husbandry and veterinary preventive medicine; and the National Agricultural Research Bureau, for insect control work. Local fellowships in China granted by the Foundation numbered 140.

CORRESPONDENCE

PREJUDICE AGAINST VACCINATION TO CONTROL SMALLPOX

Editor, *New England Journal of Medicine*,

In the issue of June 25, page 1322, there are some inaccuracies, the most glaring being that Dr. Boylston was the target of a bomb. The bomb was thrown into the house of *Cotton Mather*, who was the instigator of the inoculation for smallpox.

I wrote an article covering inoculation but it was

too long for *The New England Journal of Medicine.*

Yours truly,

STEPHEN H. BLODGETT, M.D.

South Lincoln,
Massachusetts.

Dr. Blodgett is entirely right; the bomb was thrown into the house of Cotton Mather. The rabble, with halters in their hands, merely threatened to hang Dr. Boylston, if they could find him, to the nearest tree.

DWIGHT O'HARA, M.D.

5 Bay State Road,
Boston, Mass.

RECENT DEATH

McCARTHY—EUGENE JUSTIN MCCARTHY, M.D., of 124 Cedar Street, Malden, Massachusetts, died at the Soldiers' Home, Chelsea, July 28, 1936.

Dr. McCarthy was born in Malden and graduated from the Tufts College Medical School in 1905 and was a Fellow of the Massachusetts Medical Society and the American Medical Association. He served for fourteen years on the staff of the Veterans' Bureau in Boston and for sixteen years on the Malden Park Commission.

NOTICES

FIRST INTERNATIONAL CONGRESS OF SANATORIA AND PRIVATE NURSING HOMES

The First International Congress of Sanatoria and Private Nursing Homes will be held in Budapest from September 16 to September 21 in the rooms of the Academy of Sciences. The Secretary of the Congress reports that many prominent persons have indicated their willingness to give lectures. It is requested that any who wish to submit titles of lectures, and have not already done so, send them as promptly as possible to the Organization Committee of the Congress: Budapest Szt. Margitsziget Szanatorium Prof. dr. Benczur.

AN OMISSION

In the report of the exercises of the Annual Meeting of the Massachusetts Medical Society, the name of Dr. Peer P. Johnson as a guest of the President at the head banquet table did not appear.

Dr. Johnson was present.

REMOVAL

LOUIS ARKIN, M.D., announces the removal of his offices from 471 Commonwealth Avenue to 395 Commonwealth Avenue, Boston. Telephone: Kenmore 8100.

REMOVAL OF OFFICES

The Boston Council of Social Agencies (including Bureau of Research and Studies, Volunteer Service Bureau and Purchasing Bureau), the Boston Health League and the Hospital Council of Boston moved their offices on July 25 to those of the Community Federation of Boston on the eleventh floor of the Chamber of Commerce Building. The new address and telephone number are Room 1104, 80 Federal Street, Boston, LIBerty 8515.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, AUGUST 10, 1936

Saturday, August 15—

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Reginald Fitz.

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*Open to the medical profession.

August 24-29—Harvard University Tercentenary Celebration. See page 1166, issue of June 4.

September 4 to 8—First Congress of the Austrian Society for the Study of Roentgenology. For details address: Allgemeines Krankenhaus, Alserstrasse 4, Wien, IX, before September 1.

September 7-10—International Union against Tuberculosis. See page 554, issue of March 12.

September 7-11—American Congress of Physical Therapy will meet at the Waldorf-Astoria, New York City. See page 52, issue of July 2.

September 9 to 12—Sixth Congress of the International Society for Urology. For details address Dr. Theodor Hryntschak, Rathausstrasse 3, Wien, I.

September 14 and 15—Tercentenary Session of the Harvard Medical School. See page 1166, issue of June 4.

September 16-21—First International Congress of Sanatoria and Private Nursing Homes. See page 803, issue of April 16, and notice elsewhere on this page.

September 22, 23, 24—Twelfth Clinical Congress of the Connecticut State Medical Society. See page 217.

October 12-18—Third International Congress on Malaria. See page 1076, issue of May 21.

October 19-23—Clinical Congress of the American College of Surgeons. See page 180, issue of January 23.

October 19-31—1936 Graduate Fortnight of the New York Academy of Medicine. See page 1221, issue of June 11.

October 20-22—Academy of Physical Medicine, Annual Meeting, Hotel Statler, Boston.

October 20-23—The American Public Health Association. See page 1226, issue of June 11.

March 30 - April 2, 1937—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 21-24, 1937—American Society for Experimental Pathology. See page 1075, issue of May 21.

BOOK REVIEW

Transactions of the American Gynecological Society. Volume 60. For the year 1935. Edited by Otto H. Schwarz. 353 pp. St. Louis: The C. V. Mosby Company.

This sixtieth volume of transactions presents the proceedings of the Society at its annual meeting in Virginia on May 27, 28, 29, 1935. Its chief interest lies in the twenty-one original papers read. Notable among these are two by Mr. Victor Bonney of London, on the treatment of carcinoma of the cervix by Wertheim's operation (illustrated by a series of nineteen admirable cuts), and on the extended scope of myomectomy in the treatment of uterine fibroids. Boston readers will also be particularly interested in Dr. Louis E. Phaneuf's paper on the place of colpectomy in the treatment of uterine and vaginal prolapse; and in Dr. Newell's excellent obituary of Dr. Walter L. Burrage.